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ADDRESSES AND ORIGINAL ARTICLES

THE MECHANISM OF INFLATION OF THE LUNGS

AND THE INFLUENCE OF DEFLATION ON POST-OPERATION COMPLICATIONS.

By Sir Charlton Briscoe, Bt., M.D.,
F.R.C.P. Lond.,
PHYSICIAN TO KING'S COLLEGE HOSPITAL.

In the past three years a number of papers have appeared dealing with post-operative collapse of the lung, but the reader will find no agreed opinion about the production of this condition or the manner in which it should be treated. Nor does it appear to me that there is any clear distinction of the various conditions which are found and which have been called collapse. At least four have been included under this term, namely:—

(1) A condition in which the lower part of the lung is more or less airless, but in which there may be no symptoms. This may be called *simple deflation*.

(2) Simple deflation in which there is clearly a bronchial irritation varying in degree, with consequent mucous or muco-purulent secretion: *simple deflation with secretion*.

(3) The condition which originally attracted Pasteur's [1] attention, in which, sometimes without but generally with a very acute onset, one or both lower lobes of the lungs are found to be airless, the heart being displaced to the affected side. Purulent secretion may or may not develop: *massive collapse attack*.

(4) A condition of deflation of the whole of one lung, most commonly found in injuries (often trivial) of the chest, and not necessarily or usually as a post-operative complication. This has been fully described by Rose Bradford [2] from his experience in France during the war, and may be termed *massive deflation of one lung*.

The types of simple deflation are very common, but they have been neglected, attention having centred on the two rare conditions and their possible explanation. I hope to show that simple deflation is not merely very frequent but that it is essential that it should be studied and understood before speculation is indulged in concerning the nature of the more rare massive collapse attacks or deflation conditions.

BASIC ANATOMICAL AND PHYSIOLOGICAL FACTS.

There are two normal anatomical and physiological facts which must be kept persistently in mind, since they are constant influences in the production of deflation.

(1) *Elasticity of lung.*—Throughout the whole of life, after the first few breaths, the lungs never cease to try to contract down to an airless condition. They are prevented from doing so by the facts that they are enclosed in the thoracic cage, and that nature abhors a vacuum. The cage, however, nowhere quite rigid, shows different degrees of mobility in different parts, and such mobility is affected in various ways by different conditions. To put it another way: the lungs always deflate as much as they are allowed to, but this allowance is subject to very wide variations.

(2) *Minimal use at rest.*—Man is endowed with much more respiratory air space than he requires when at rest. This well-known and obvious fact, akin to the reserve power of the heart, has not been sufficiently stressed; to all intents and purposes it has been ignored. This is accounted for by the fact that deflation has only been discovered when some pathological condition has necessitated examination, and hence a pathological condition has promptly been invoked as the cause of the deflation. The true sequence in these cases is that deflation ensues as a simple consequence of rest in bed.

As illustrating this point, the following three papers are of importance. My colleague, Dr. J. L. Livingstone,[3] has shown that, when changing from upright to supine, the volume of the chest, as measured by the excess of air exhaled, is reduced by about 500 to 1000 c.cm.—10 per cent. to 20 per cent. of the total volume of the lungs—and that in the intermediate position of reclining at an angle of 45° the diminution is proportional—i.e., about one-half. This reduction occurs within ten breaths. W. H. Wilson [4] has shown in a similar manner that the reserve air is reduced under similar circumstances by about 1200 c.cm. His observations were made two minutes after the change from the erect to the supine. These two observations were made on normal individuals.

J. R. Head [5] has shown that the vital capacity of the thorax is markedly diminished—from 8 per cent. to 80 per cent.—when tested during a period of from 2 to 18 days after operations, such as those on the abdomen, for removal of the breast, and for the treatment of hæmorrhoids. The value of this paper would be enhanced if the author could have recorded the diminution in the vital capacity of individuals who were rested in bed without operation—i.e., if he could have tested what occurs in the normal.

These three investigations show beyond doubt that under the circumstances in which the observations were made a considerable reduction in the volume of the thorax takes place. Livingstone's observations were made, so far as possible, with involuntary or unconscious—as opposed to forced—respiration; the other two observations were made with forced, and therefore conscious, expiration; they are of less value when applied to the usual and unconscious respiration, but are confirmatory of that change which Livingstone records in the normal individual. From these three papers, however, a logical deduction can be made: that on assuming the supine posture a diminution in the intrathoracic volume takes place immediately, and that such diminution is not limited to the first ten breaths but is continued for some time. In these circumstances the volume of the lung is decreased. Such a diminution might occur equally in both upper and lower lobes, or by a diminution in either one or the other. Further, such a marked diminution in the size of one lobe, either the upper or the lower, might occur as would necessitate an over-inflation of the alternate lobe.

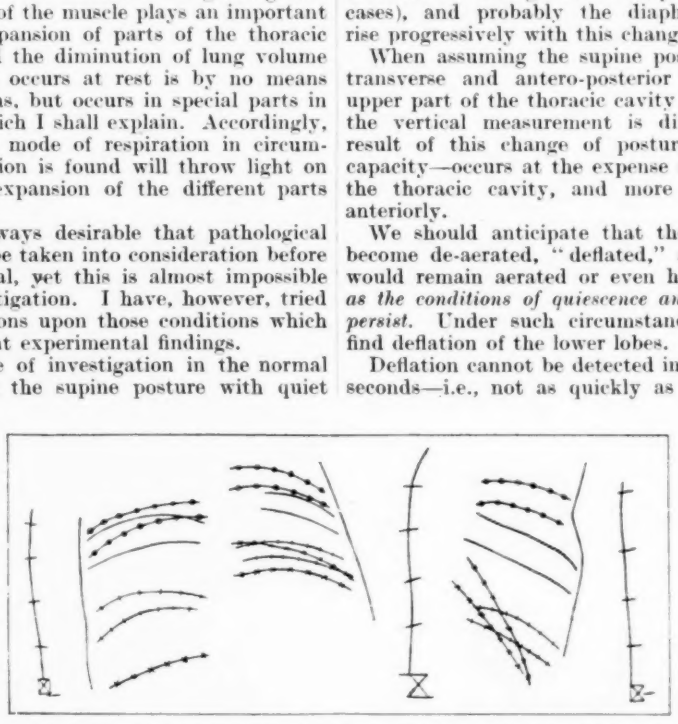
The thoracic cage is a complicated structure of bone and muscle. In the past an attempt was made experimentally to gain an insight into its movements by reproducing the effect of maximal contractions of the muscles. In life, however—except perhaps in yawning, sighing, and asphyxia—such maximal contractions are not evoked; the usual nerve impulses are not strong enough to produce them. At rest the weaker stimuli conditioned by diminished respiratory requirements evoke a smaller thoracic expansion. This diminished expansion is not the result of a general

decrease of contraction of each muscle concerned. A process of selection occurs to determine which muscle or part of muscle shall respond to the weaker nerve impulse. In this selection or grading the condition of stretch of the muscle plays an important rôle. Hence the expansion of parts of the thoracic cage is unequal, and the diminution of lung volume and deflation which occurs at rest is by no means equal in all directions, but occurs in special parts in a special manner which I shall explain. Accordingly, an inquiry into the mode of respiration in circumstances where deflation is found will throw light on the mechanism of expansion of the different parts of the lung.

Although it is always desirable that pathological findings should not be taken into consideration before establishing a normal, yet this is almost impossible in the present investigation. I have, however, tried to base the conclusions upon those conditions which most nearly represent experimental findings.

Following the line of investigation in the normal individual assuming the supine posture with quiet breathing, two important points emerge—viz., an immediate change occurs in both diaphragm and thorax.

(1) The diaphragm is elevated.[6] The usual X ray inspection (tube behind and screen in front) demonstrates this point. Especially is this seen in the mid-axillary region, but mesially to the nipple lines the elevation is less marked. This view does not give a complete picture of what happens. By the employment of tangential views occurrences immediately below the surface of the ribs at any given points can be investigated, and the height (of the point) at which the diaphragm leaves the chest wall above the costal margin—the upper level of the costophrenic sinus—can be measured. By this means it is seen that an immediate rise occurs in the level of the costal diaphragm, particularly at its posterior part, in the scapular line, averaging 4 cm. on the right and 4·5 cm. on the left. The rise is much less—2 cm.—in the nipple line. There is, therefore, when assuming the supine posture, a general rise in the level of the costal diaphragm, and this is more marked in the postero-lateral than in the anterior regions. This elevation is least marked in the antero-posterior mesial area, the plane of the crus. At the same time the natural inspiratory descent continues, although it may be diminished. Pasteur [1] has shown that this diminution of the diaphragm movement persists some days after operation.



X ray examination (tangential), showing the points at which the diaphragm leaves the chest wall in different postures. The left figure records that point in the post-scapular line on the right side. The figure to the right illustrates the same in the left post-scapular line. The central figure illustrates the points in the right nipple line. In each instance: (1) The line with circle equals supine. (2) The straight line equals reclining at an angle of 45°. (3) The line with single dash equals standing. (4) The line with stars equals knee-elbow position. Where the lines are in pairs the top one is expiration, the lower one inspiration. Where it is single the position in both phases is constant.
The cross is placed on the lowest rib in the line under investigation. Vertical cross bars placed at inch intervals lie on outer surface of chest wall. Other vertical line represents pleural surface of chest wall. Subject, a healthy medical student, aged 23. Quiet involuntary respiration throughout.

(2) In the thorax the transverse diameters (at the three levels of the manubrium, gladiolus, and lower costal margin) and the antero-posterior diameter (at the first two levels) show increases.[6] The changes in the diameters of the upper part of the thorax attain their maximum in a few hours, but the transverse diameter at the level of the lowest rib continues to increase during several days (in post-operative cases), and probably the diaphragm continues to rise progressively with this change.

When assuming the supine posture, therefore, the transverse and antero-posterior dimensions of the upper part of the thoracic cavity are increased, while the vertical measurement is diminished. The net result of this change of posture—a diminution of capacity—occurs at the expense of the lower part of the thoracic cavity, and more at the back than anteriorly.

We should anticipate that the lower lobe would become de-aerated, "deflated," and the upper lobe would remain aerated or even hyperinflated *as long as the conditions of quiescence and recumbent posture persist.* Under such circumstances we do, in fact, find deflation of the lower lobes.

Deflation cannot be detected in the course of a few seconds—i.e., not as quickly as the changes in the diameters of the chest wall or the elevation of the diaphragm. It is marked in 36 hours.

Physical Signs.

Deflation of the lower lobe in varying degree is very common. It is not detected because, in the routine examination, the first few voluntary breaths taken by the patient obliterate the signs, and the front of the chest is examined fully before that of the back is commenced. It is when the patient does not, or cannot, take the deep breaths that the signs are detected. If, however, a rule is made to percuss and auscultate the back before asking for a deep breath, signs are readily detected. The signs will be found fully developed at the end of 36 hours, or less, in any adult patient who lies quiescent and, therefore, regularly and typically for the first few days after operation.

As the lower lobe deflates, the anterior and inferior margins retreat, the base rising, and when deflation is complete the lobe occupies an area beneath a triangular space delineated by: (*a*) the middle-line spines of the vertebræ, (*b*) a horizontal line through the eighth rib in the scapular line, and (*c*) an oblique upper margin from the fifth dorsal vertebra to the horizontal line (*b*) as this cuts the posterior axillary line. This is the area where the signs are detected. Over a lobe partially deflated there are to be made out:—

(1) Diminished resonance to percussion.

(2) Diminished air entry on inspiration, tubular breathing, and sometimes crepitations.

(3) Increased voice sounds, and increased tactile vocal fremitus.

These are the signs of a lung of which the density

is increased, in this case by deflation. Where deflation is early the signs are less marked and crepitations occur with inspiration. As the lower lobe deflates more completely and shrinks towards the root of the lung and away from the chest wall, the air entry is less audible and voice sounds remain increased and bronchophonic. The triangular area contracts as the upper lobe becomes over-expanded and encroaches on it.

The diaphragm being elevated as a whole (back, front, and sides, the mesial portion least), the organs in relation to it are elevated with it. The absolute dulness below the eighth rib posteriorly is due to the elevation of the diaphragm and underlying organs. The liver dulness on the right side anteriorly is also elevated as high as the fifth rib or fourth space, and resonance from the abdominal organs may replace the lower area of normal liver dulness. On the left side the apex beat is elevated and rotated outwards so as to be behind the fifth rib or in the fourth space. The stomach resonance is high in the axilla.

When a deep diaphragmatic breath is taken, protruding the abdomen, these signs become less marked and, with repetition, disappear with a shower of crepitations, only to return with renewed quiescence. Those normal individuals with (*a*) big respiratory capacity, (*b*) a natural tendency to thoracic breathing, (*c*) signs of emotional distress, (*d*) loss of muscular control of the lower thoracic margin, (*e*) evidence of general debility, and (*f*) those who remain in such a posture that the spine is extended, tend to breathe with the upper thorax at the expense of the diaphragm and so to develop deflation of lower lobes the more readily.

The two first conditions are relatively commoner in the young, and in them deflation occurs the more readily. Collapse attacks of extreme severity are many times more frequent in the young than the old.

Pathological Evidence.

(1) *Simple picture.*—This appearance of simple deflation of the lungs has escaped attention, largely owing to the fact that in a routine post mortem we do not obstruct the trachea before opening the thoracic cavity. In consequence air escapes from the lungs and ante-mortem deflation is less noticeable. When the trachea is properly blocked—as by ligature over a cork inserted through a tracheotomy incision—the relative size of the lobes is readily noticed.

The simple picture is as follows: the deflated area is smaller than normal, darker in colour, and more solid to the touch, and the capsule is wrinkled. It contains little air, and therefore tends to sink in water. Microscopically, the walls of the air vesicles are approximated, blood-vessels are dilated, and fibrous tissue is distended and more obvious than usual. The fibrous tissue has to stretch to follow the shrinkage due to deflation. The walls of smaller bronchioles are sometimes as much approximated as those of the still finer portions of the lungs. The larger bronchial tubes are patent, and may or may not contain some mucus and cells. In a simple case there is no evidence of pleurisy or of inflammatory action in the lung or of desquamation of bronchial mucous membrane. The condition may be found affecting parts of the lower lobes in different areas and degrees; it is not of necessity limited to the lower lobe. The posterior part of the upper lobe may be deflated. All stages of incomplete deflation may naturally be found, and differences in the march or mode of the deflation of the two lungs are detectable.

This is the condition which is obviously present during life in many of our "bed patients" in greater or lesser degree. Ante-mortem œdema obliterates the evidence and, naturally, apart from œdema, the whole picture is frequently modified by the existence of injury or disease. A careful study of selected instances will help to illustrate. Such instances arise when death has occurred suddenly and without struggle, or when terminal œdema has not occurred in those who have lain quiescent prior to death.

The simplest condition may be best seen under the following circumstances, many examples of which have been observed and studied:—

(*a*) *Coma cases*, in those who have been unconscious and have lain still for hours or days prior to death as in cases of cerebral tumour. During life recumbency has been absolute, and there has been no forced breathing. In these there has been no impediment to the diaphragm or to the chest wall. There has been increased respiratory movement of the upper chest, and diminished abdominal protrusion. The movement of the epigastrium may even be reversed—inspiratory recession. By X ray the diaphragm is seen to be high and to descend in inspiration. The lower lobes are found post mortem in the condition described above. There is no obstruction to the bronchial tree.

(*b*) *Operation cases.*—During the first few days after operation, especially if there has been an incision through the abdominal wall, the patient is exhausted and movement painful. The epigastrium is found to protrude to some extent in inspiration, showing clearly that the diaphragm is not paralysed. The upper thorax is hyperactive in inspiration. The diaphragm is high, but inspiratory descent, though present, is diminished. When cases of this nature die suddenly and unexpectedly within the first few days after operation, as from pulmonary embolus, there will be found marked deflation of the lower lobes, with hyperinflation of the upper lobes. There is no obstruction to the bronchi.

(*c*) *Spinal cord lesions.*—In conditions of spinal cord obstruction in the upper dorsal or lower cervical region the patient is most comfortable when propped up, for gravity helps to counteract the notable elevation of the thorax by the scalene in inspiration and thus assists expiration. The intercostal and abdominal muscles are paralysed. The diaphragm can be seen by X ray to be at a remarkably high level and moving with inspiratory and expiratory excursion. (By volitional effort it can be temporarily depressed to a greater degree than is otherwise seen.) The lower lobes are more completely deflated than in any other condition. Sometimes the deflation extends to the back part of the upper lobes. The upper lobes are markedly over-distended. In cases of some weeks' or months' standing the bronchi contain a little material which, on investigation, is found to consist of cells from which the nuclei have mainly disappeared, and a small amount of mucus. There is no obstruction to the bronchi. The physical signs of deflation of the lower lobes are readily detected in such cases.

(*d*) *Unilateral phrenic paralysis.*[7]—In conditions in which one phrenic nerve is blocked by pressure, as from glands in the neck, thus throwing out of action half the diaphragm, the movement of the chest wall is not impeded, but may be increased. The unaffected portion of diaphragm contracts normally; the paralysed half is somewhat elevated and has a paradoxical action. The lungs show (*a*) a considerable degree of deflation of the lower lobe on the affected side, (*b*) a fully inflated lower lobe on the sound side, (*c*) a considerable degree of deflation of the upper lobes, more on the affected than on the

unaffected side. There is no obstruction to the bronchi of either the lower or the upper lobes.

The above four instances, being concerned with normal lung, are examples almost experimentally ideal. They show that under conditions of respiratory activity reduced to the minimum by posture or weakness, a defect of expansion of the lower lobe occurs. When correlated one constant factor emerges: the lateral part of the diaphragm beneath the deflated lower lobe or lobes is elevated. The condition of the chest wall varies, as does the inspiratory descent of the diaphragm. We are led to the conclusion that expansion of the lower lobe requires a combination of two factors: (1) a low, and (2) a contracting diaphragm. Applying the same interpretations, the degree of inflation of the upper lobe is dependent (1) upon the maintenance of a condition of postural tonus in the whole diaphragm (as opposed to paralytic tonelessness in part); and (2) upon activity of the upper chest. This latter deduction is in accord with the findings in the oft-quoted case of Pearson Irvine.[8]

So far we have considered conditions marked, as far as the lung is concerned, by nothing but simple lack of muscle movement. Further confirmation is provided by examples of pathological conditions, for instance:—

(*e*) *Cardiac dilatation*, when associated with pericarditis, is recognised as a condition in which deflation of one or both lower lobes occurs. The so-called rheumatic lung [9] is an example of deflation with an avirulent infection. I have no X ray evidence to show that the diaphragm is elevated. The physical signs—raised apex beat and liver dulness, with diminished epigastric protrusion and excessive thoracic breathing—indicate elevation. But further, in cases of cardiac dilatation due to valvular lesions, with crepitations (so-called œdema) of the bases, when sudden death occurs—as by rupture of an aneurysm into the pericardium—the lower lobes show the typical appearance of deflation and no œdema is present. There is no obstruction to bronchi. Respiration is preponderatingly thoracic. Livingstone and I have shown experimentally in rabbits that certain interferences with the heart through the open pericardial sac causes an elevation of the diaphragm. So-called "back-pressure lungs" are, in my opinion, merely deflated, and œdema, as in other conditions, is only a terminal event.

(*f*) *In acute pleurisy*, affecting one lower lobe, the homolateral costal diaphragm is raised and movement is diminished. In the early days the upper chest is held elevated, and the lower chest is fixed by intercostal and abdominal muscles. In cases of pleuropneumonia in which death occurs during the stage of red hepatisation, as the result of the pleurisy the lower lobe on the affected side is found actually to be smaller than its fellow—i.e., in the early days it is partially deflated. The main bronchi are not obstructed.

(*g*) *In localised peritonitis*—as, for example, that affecting the peritoneum in the region of the right lobe of the liver—an elevation of that half of the diaphragm, with diminished movement, is found by X ray. The restricted respiration is carried out by elevation of the chest—scalene action—and upper thorax expansion, deflation of the corresponding lower lobe being marked.

(*h*) *In cases of diphtheria* with paralysis of the diaphragm the condition of the lungs described by Pasteur [10] is one of deflation on which œdema is frequently superadded. In these patients the supine posture is adopted.

SUMMARY.

The foregoing observations show that whenever the respiratory effort is at a minimum, and the individual in a reclining or supine posture, the capacity of the chest diminishes. As soon as these conditions arise, the lung commences to deflate, this deflation being mainly seen in the lower lobes and being independent of bronchial obstruction. Toxæmia—or other conditions producing lowered vitality—exerts an effect through muscular weakness and greater passiveness of the subject, while local conditions influencing the height of the diaphragm further accentuate the deflation.

In adults, deflation is marked in the course of 36 hours; in children it is detectable within half an hour. The signs can disappear readily if the patient be induced to take deep diaphragmatic inspirations. When quiescence is resumed, deflation promptly recurs. It is more accentuated when the costal margin is unstable as the result of lack of action of the abdominal muscles. This defect is especially marked when the muscle has been injured, as during operation. The condition is typically found in people who remain in one posture because they are very ill or physically weak, or because pain prevents movement. The fundamental cause is the reduction of the necessity for a large air entry, and it is not necessary to postulate any other causal factor. The point to be accentuated is that deflation of the lower lobe occurs in all individuals who recline in one position without exerting themselves. If the lung is temporarily expanded or inflated deflation recurs at once on resuming quiescence.

The Influence of Deflation on Post-operative Conditions.

When pulmonary complications follow operation, deflation must be regarded as the primary condition on which further changes which occur in the lower lobes are implanted, and no explanation of pathological changes can be complete or accurate which does not take this deflation into account.

SIMPLE DEFLATION.

A lung deflated as described possesses a circulation, and the activity dependent thereon continues—e.g., in the nutrition of the lung in the function of reproduction of the cells lining the alveoli and of the bronchi, in the function of the mucous glands in producing mucus in varying quantity, and in the general response to infection. With little irritation this will produce no clinical manifestation, so we find:—

(1) *Simple deflation.*—In most instances this is not detected. No symptoms occur to attract attention. It merges into:—

(2) *Simple deflation with secretion:* (*a*) *Non-pathogenic.*—Sometimes a loose, shallow, unproductive and frequent cough occurs, suggesting a liquid mucus which is not brought up to the mouth. It resembles the typical cough in the case of an empyema. Frequently in uncomplicated cases of deflation, as after operation, the mucus is expectorated at the end of three to seven days. It may be grey in colour, or green or dull red, not giving a test for blood. Besides mucus and shadow alveolar and bronchial cells, a number of bacteria are present which, in the absence of polymorphonuclear leucocytes and pyrexia, are clearly acting not pathogenically but as parasites or saprophytes, and they explain the colour of the mucus. In some cases there is a tendency to the secretion of an excess of mucus. The secretion may vary in consistence from that of a bronchorrhœa

to that almost resembling fibrinous bronchitis, but such extremes are rare.

(*b*) *Pathogenic.*—If the bacteria act pathogenically the secretion becomes purulent, and pyrexia and signs of bronchitis with distress on coughing result. Inhalation of infected material into the deflated lung consequent on deeper respiratory movements gives rise to various conditions of bronchitis, bronchiolitis, broncho-pneumonia or pleurisy. In such instances pleurisy is not very acute. The bronchus may be infected before, during, or after the operation. These two types are readily understood.

THE MASSIVE PULMONARY COLLAPSE ATTACK.

This type was originally described by Pasteur.[1] Usually, though not invariably, the onset is very acute, with pain, orthopnœa, cyanosis, and general distress. These symptoms, however, pass off in 48 hours. On the affected side the lower lobe of the lung is found to be airless. Although the upper lobe is over-expanded the air entry is poor. The heart is displaced toward the affected side, which, on measurement, is found to be diminished. On the unaffected side the lung is over-distended and the air-entry is good. If both sides are affected, the heart is mesial. Purulent secretion may or may not develop. The genesis of this condition is obscure, and three main theories have been put forward to explain it, none of which take cognisance of the condition of deflation which exists in the early days after operation, and therefore prior to the attack.

1. The first theory is the original theory of Pasteur,[1] which ascribed the condition to sudden paralysis, which might be reflex in origin, of some of the respiratory muscles. He clearly likened the condition of these to the paralysis of other muscles which is found in diphtheria, and ascribed the various changes directly to a sudden lack of distending power.

2. The second theory is that the condition is the direct result of obstruction to the bronchi. This obstruction may be due to mucus or to spasm or paralysis of the bronchial muscles. The theory is based upon the well-known experiments of Lichtheim[11] and the results of observations on foreign substance in the bronchial tubes. In most cases the holders of this theory—Elliott and Dingley,[12] Boland and Sheret,[13] Santé and Morrison[14]—would not admit the rôle of respiratory muscular defect; while others (Lee,[15] and Jackson and Lee[16]) would allow that embarrassed respiration may play a part. Woodburn Morrison[17] is driven to invoke the necessity of vagal or sympathetic action.

3. The third theory suggests pulmonary emboli as a causative factor (Cutler and Hunt[18]), such emboli being held to be not only of venous origin, but travelling also through the lymphatics. By both paths the origin of the emboli is placed in the neighbourhood of the wound, and in many cases sepsis is admitted as a contributory factor.

Each of these theories evades recognition of the physiological condition of the lungs found in the resting patient in the early days after operation, but tacitly assumes that the lungs are in the naturally expanded condition found in ordinary life, and accordingly seeks to explain the sudden change from such a state into that found in the massive collapse attack. This false premise renders criticism of these theories almost unnecessary and certainly difficult. The points requiring explanation are the suddenness of the attack, its duration, the pain and pyrexia, as well as the relationship of the physical condition of the lungs and heart found at the moment to the condition of deflation of the lower lobes present before the attack.

Theory 1.—In his post-diphtheritic cases Pasteur[10] used the term paralysis to indicate a condition of the diaphragm similar to the paralysis of the eye or pharyngeal muscles. Basing the condition of the post-operative massive collapse attack on the foundation of paralysis of muscles in diphtheria, he considered that the condition of the lung was a similar development to that found in diphtheria, but that, instead of occurring gradually, the onset came with cataclysmic suddenness. When limited to one lobe this was due to paralysis of one phrenic nerve and of half of the diaphragm.

Except in cases of direct pressure on or injury to the nerve, I have never seen true phrenic paralysis. Unilateral phrenic paralysis, when uncomplicated, gives rise to different signs.[7] Further and more important, as underlying the pathology of the collapse attack, in these cases of post-diphtheritic paralysis so-called), the diaphragm can be induced to contract. Physical signs show that it is elevated, but when the lower thoracic expansion is controlled—depressing the lower ribs by axillary pressure and bringing them towards the middle line—the diaphragm contracts, the epigastrium is protruded, and the signs of collapsed lung disappear. In ten cases of this condition I have never failed to demonstrate this point, a fact which shows that in these post-diphtheritic cases the condition is really one of marked postural deflation. I agree that a physiological lack of distending muscular power is the basis of deflation, but I do not agree on the mechanism by which it is suggested that the acute collapse attack is produced. Further, pain and pyrexia are not explained.

Theory 2.—Deflation certainly occurs without endobronchial contents. This fact is against the theory which explains the condition as analogous to that of a foreign body in a large bronchus. It is difficult to see how a large bronchus can be occluded by muscle spasm. Occlusion could occur in a small bronchus, and such action might be regarded as a protective mechanism for the part of the lung rendered airless. The theory does not explain (1) the constancy of the deflation in the lower lobe, (2) the irregularity of its distribution in the lower lobe, (3) its extension in the upper lobe on rare occasions, in each instance without symptoms or bronchial obstruction, or (4) the fact that what I have termed deflation with secretion occurs, and without these acute symptoms. It is possible that a spasm of a bronchiole muscle can withstand the depressive power of an active diaphragm; if the spasm could do so it is difficult to conceive that with a contracting diaphragm the imprisoned air could be absorbed. Nor does it explain the rapid expansion of the lower lobes which occurs when diaphragmatic movements are induced. Still less does it explain the suddenness of the onset of massive collapse or the pyrexia or pain.

Theory 3.—Deflation occurs without evidence of emboli. Such limitation of distribution does not occur in other conditions in which emboli invade the lung.

None of these three theories takes cognizance of the fact that the massive collapse attack comes on as a rule a day or so after the operation, and is therefore implanted on the condition of the lung existing at the moment. This I have shown to be one of deflation affecting both lower lobes, and without temperature or pain. The attack is associated with pyrexia, indicating infection, and acute pain. As a rule the onset is sudden and unexpected. Occasionally a sensation of something impending is present. The affected side is small below, expanded above and motionless, and air entry is deficient.

In my opinion these massive pulmonary collapse attacks are really instances of acute pleurisy occurring when the lower lobes of the lungs are already in a condition of simple deflation, and my reasons for this opinion are as follows:—

(1) In many of the instances described the presence of pleurisy has been admitted, either in the account of cases which have recovered, or in that of the post mortem. (Though admitted to be present, it has been excluded as the cause of the attack.)

(2) Muscles acting on the affected side of the chest behave in the same manner as they do in cases of pleurisy—i.e., (*a*) the lower intercostals are firmly contracted, (*b*) the scalenes and upper intercostals are contracted also, but to a lesser degree, in the early stages, (*c*) the abdominal muscles acting on the lower thoracic margin are similarly involved.

(3) These lower intercostal muscles are intensely sensitive to pressure, which increases the pain exactly as in pleurisy from other conditions—e.g., pneumonia. The abdominal muscle, which has been incised at operation, when forced into a protective contraction is intensely painful. (Generally in pleurisy the abdominal muscles are not sensitive to pressure.)

(4) The sudden onset of pain corresponds to the similar onset in many cases of pleurisy, and in both conditions may be first noticed following a movement.

(5) The duration of the acute pain is about 36–48 hours, which is also a common time for the duration of pain of acute pleurisy.

(6) Relief in both pleurisy and the collapse attack is immediate when suitable restriction, as by a tape and spring, is applied. Mechanical restriction of chest movement, by performing the work of the intercostal and abdominal muscles, allows these to relax, hence the relief of pain.

(7) Pleurisy explains the temperature as well as the physical signs—i.e., (*a*) tonic contraction of intercostals causing a diminution in the size of the lower chest; (*b*) tonic contraction of the elevated costal diaphragm on the affected side; (*c*) persistence of a state of deflation already present before the attack; (*d*) deficient air entry resulting from imperfect expansion of the over-distended upper lobe (it remains over-inflated, though silent, when the scalene and upper intercostals are tonic, but deflates in those cases in which, a day or so later, the upper intercostals go out of action); (*e*) compensatory over-expansion of the other lung. (*b*) and (*c*) explain the shift of the mediastinum to the affected side.

The essential difference between a massive collapse attack caused by pleurisy and an ordinary pleuritic attack lies in the facts that in the former the lower lobe is already deflated, and in many cases a muscle in which tonic contraction occurs has been incised, and contraction is painful. The uncertain point here is the assumption of a tonic or spastic condition of the costal diaphragm, as a result of pleurisy. I think it justified because I find the same changes (elevation and lack of motion) in the diaphragm in these cases of collapse as are found in acknowledged cases of pleurisy. The infection has in most cases come through the diaphragm from below, and I am ascribing to the diaphragm a reaction which certainly occurs in other muscles under like circumstances.

Hence I consider that these cases described by Pasteur as massive collapse attack of the lungs are instances of well-marked simple deflation in which the condition on one or both sides is suddenly modified by the onset of an acute pleurisy. I do not believe that bronchial obstruction or embolism have anything to do with them.

DEFLATION OF A WHOLE LUNG.

The explanation of deflation of a whole lung is more difficult because the condition is seldom seen in civil practice, and is not essentially a post-operative condition. Under war conditions slight or severe injuries to the chest were exciting causes, producing deflation of the whole lung on the same side as the trauma, or on the opposite side. This injury might be to the upper or lower part of the chest wall. It raises questions of a reflex inhibition of expansion and of injury by contra-coup. (In many experiments to elicit a reflex inhibition of one-half of the diaphragm only very occasionally was evidence obtained at all, and then only by a reflex arising from the chest wall on the application of heat or cold. The effect was slight and incommensurate with the stimulus.) In two cases of chest injury seen recently, the one severe and the other slight, both in children, the whole of the left lung was out of action—deflated. The diaphragm was high and the outline not clear, so that immobility was not definite. The scalenes and upper intercostals on the left were toneless, giving rise to a flattened and motionless upper chest, while the lower intercostals were tonic. Both the upper and lower areas of the chest were thus diminished. In these two cases the mechanism of production clearly was defective muscular action.

This brings all four types of deflation into one category—deficiency of muscular action in expanding the lungs. At present we must consider the fourth condition as differing from the first three, inasmuch as the upper part of the thorax is out of action, the whole lung being affected.

Therapeutic Indications.

The conclusion to which we have been led is that, whenever an individual remains at rest in one posture with respiratory exchange at a minimum, deflation is bound to occur. Youth, early adult life, certain types of breathing and conditions of disease producing marked weakness or unconsciousness, naturally increase the tendency to this condition. If the individual is disturbed, the expansion of the lung may be partially or fully restored for the time being, only to deflate when the disturbance is discontinued. Local lesions, injury or disease may modify the distribution or extent of the deflation, and the clinical picture may be altered by complications occurring when the deflation already exists, but these are addenda and should not be allowed to disturb our conception of the basal physiology of the condition—a deflation of the lung resulting from diminished muscle action. The more severe the operation, especially in the upper region of the abdomen, the more marked will be the deflation.

The occurrence of massive collapse attacks has suggested that therapeutic measures should be applied to every abdominal operation case, in order to obviate post-operative pulmonary conditions. Among such routine measures are the administration of carbon dioxide to produce deep inspirations, and turning the patient on his side, with or without smacking. Any routine treatment is a mistake, because so many escape any symptom at all, and to put patients to pain and distress within a few days of the operation is certainly unnecessary and inexpedient. It is not advisable to promote deep breathing if there is a likelihood of carrying infection to the deeper part of the lung.

Simple deflation.—So long as the individual is quiescent, only requiring the employment of a small portion of his respiratory area, there is no need for

the lung to be fully expanded, or for muscular action causing pain to be encouraged. More lung will become inflated as more respiratory changes are necessitated. The majority of subjects of operation pass through these stages without symptoms which attract attention. It is therefore unnecessary, to say the least, to adopt any routine measures which aim at promoting once or twice a day expansion of the lung accompanied by fatigue and distress.

Simple deflation with secretion.—During the course of the administration of a general anæsthetic, respiratory movements of the greatest intensity are induced, and all parts of the lung must be over-distended. At this time there is every opportunity for infective material to be aspirated. After the operation the breathing becomes as shallow as possible. As a result, an excess of mucus may be secreted in the large bronchi. In many cases this is not pathogenic. There can be no object in causing it to be inhaled into smaller tubes. If the respiratory tract has become infected, either in the main bronchial tubes or below the level of the cough reflex, no method of fully expanding the lung is going to improve matters. Sputum is viscid, coughing painful, and every effort exhausting. The lung requires time to develop resistances, general and local: serum, leucocytes, alveolar cells, and mucus. The condition may be made worse if the infection is aspirated to the very surface of the lung, and pleurisy results. Further, when therapy to expand the lung has been employed, directly it is discontinued the lung proceeds to deflate, a progress only broken by the cough and the next therapeutic application.

The massive collapse attack, in my opinion, is the result of pleurisy, which may arise by a spread to the surface from the deeper parts of the lung through the visceral pleura, but which more usually (where no pulmonary symptoms have preceded) originates from sepsis arising from the seat of operation through the parietal pleura. Forced expansion will not improve this. It is important to see that full respiratory activity has been reacquired by the subject after operation, especially if the lungs have been involved, but this should not be investigated until 14 days or so after operation, or when the patient is getting up.

I therefore strongly deprecate methods which aim at expanding the deflated lung in the early stages after operation. In the great majority of cases no effect of the deflation is perceptible, and the means suggested are unpleasant; where symptoms arise the procedure on theoretical grounds is inexpedient, and a paper by Dr. Fuller [19] confirms that practically it is detrimental.

Points to be recommended are:—

(1) After operation the sitting-up posture assists respiration because gravity helps expiration by allowing the thorax to descend, counteracting the inspiratory elevation of the scalenes and intercostals and obviating the necessity for abdominal muscle action for this purpose. This is especially true of the type with thoracic breathing. The individual should be allowed to choose the position which is comfortable, and be encouraged to change frequently.

(2) Bandages should not be too tight above the level of the umbilicus. It is here that abdominal protrusion in diaphragmatic inspiration is greatest in degree. On the other hand, a tight bandage supports the incised muscle. It is largely a question for the individual patient and the type of breathing he has developed.

(3) Movements of head and arm and of all sorts, as soon as these no longer cause pain, should be encouraged as inducing contraction in abdominal muscles and so relieving muscle monotony. The contraction of the diaphragm is increased, its level is lowered, and a step is taken towards expansion of the lower lobes.

(4) When non-viscid sputum is present: (*a*) the mildest symptom is half-hearted cough 18 hours or more after operation, sometimes as much as every half-minute, with no expectoration. Eventually some mucus is expectorated, usually after four or five days. This has accumulated in the "test-tube bronchi." There is not enough energy to get air behind it. As a rule, this requires no treatment. (*b*) If the condition is troublesome, the patient should be instructed to breathe in as slowly and deeply as possible and then to give a short, sharp expiration, which produces expectoration. During this effort he should support the lower thorax with a hand in either lower axilla, the nurse supporting the abdomen with her hand on the bandage over the area of incision. (*c*) After the third or fourth day, in addition, he may try gently turning into such a position that gravity helps the mucus to run out of the bronchus into the trachea, where it can be brought up. (*d*) If pyrexia is present and the sputum is purulent instead of mucoid, a Burney Yeo inhaler should be employed with the usual antiseptic inhaling fluid, the inhaler to be worn as continuously as possible. The success of this is dependent on the manner in which it is introduced, on the instruction that deep breathing is contra-indicated, and on seeing that the face is not burned by the creosote.

(5) Should the temperature rise after the lapse of 24–48 hours with respiratory distress, the sputum will at first be scanty and viscid, and the cough irritating and non-productive. Treatment should chiefly be directed to rendering sputum liquid and easily expectorated. Most important is the rapid administration of alkalis well diluted, every hour or two hours until the urine is alkaline, and of medicated vapour inhalants and such anodynes as are safe. In the common type of condition, beginning on the second or third day, the temperature will be down and expectoration easy, though the cough is still painful, on the fourth or fifth day.

(6) When more severe, the infection may be spread to deeper parts. The cough and associated deeper respirations expand the lung and fresh areas are infected. Hence arise bronchiolitis, broncho-pneumonia, pneumonia, and late patchy pleurisy, with the usual sequelæ. Treatment is such as is usually employed for these conditions. I have not met with cases of such severity as to require that the bronchial contents should be removed by mechanical means. The upper lobes alone are quite capable of coping with the respiratory requirements when at rest.

The distress of the collapse attack, as stated above, is readily relieved in a few minutes by controlling the movement of the side affected, as is the pain in pleurisy. A broad tape with a spring inserted is most efficient. The pain on coughing is not extinguished in either of these conditions, and for the same reason. A septic pleural effusion with its consequences may, of course, develop in pleurisy following operations, as in other forms of pleurisy, and requires similar treatment.

PROPHYLAXIS.

The troublesome time for the development of pulmonary infections is during the earlier days after operation. The longer the interval the greater the recovery from shock, asthenia, disinclination to move, and pain on so doing, and consequently from the deflation in the lung present in the earlier period.

The infection may have spread from a chronic condition in the patient's respiratory tract or mouth, when, generally, it does not lead to very severe results, the patient being no doubt to some degree protected. The greater the care taken to eradicate sepsis in these areas before operation the better. Probably the more severe cases arise from operation when the subject is starting a cold; I have seen many instances of this. The pulse chart shows an increase of rate within the 36 hours and the temperature chart a slight rise within the 24 hours preceding the operation, and the patient has admitted that he was not well but has thought the cause was fear. Except in cases of emergency, if there is a suggestion of acute respiratory indisposition, the operation should be postponed, though such a course is upsetting to all concerned. One of the worst cases of post-operative pulmonary infection I have seen arose after an operation on a quiescent appendix when the patient was developing a cold and had a temperature of 99·2° F. It is widely recognised that pulmonary complications in the majority of cases follow within 48 hours of the operation; 48 hours or less is a common incubation period in ordinary life for the development of catarrhal symptoms after exposure to infection. There is therefore every reason to surmise that signs and symptoms occurring 24–48 hours after the operation are attributable to infection about that time. Hence prophylaxis against infection at the operation is a most important point. In addition to the lowering of reflex activity by morphia, the drying-up of secretion by atropine, the suppression of the naso-pharyngeal filter by a mouth gag, and the excitation by the anæsthetic of very deep inspiratory efforts in a moist superheated atmosphere, there is considerable overcrowding in the immediate vicinity of the patient.

Of four patients operated upon in one afternoon, three developed acute respiratory complications and one died. A member of the operating team was developing an acute cold. Among the populace there seems to be an idea, half believed by the medical profession itself, that the infections of doctors and nurses differ from those of other people and are not communicable. Liability to infection from the surgical team, from ward nurses in attendance, and from visitors ought to be avoided. Face masks are insufficient protection in such conditions. Persons with acute infections should not be allowed near the patient during the early days after operation.

CONVALESCENCE.

It is just as important to see that full respiratory movements are restored after any abdominal operation as it is after pleuritic affections. There is the greater tendency for the individual who inclines to thoracic breathing to neglect fully to re-employ the diaphragm. As a rule, this defect rights itself as exertion increases; until it does so complaints indicative of dyspnœa, asthenia or lethargy, and of pain about the sternum, are apt to be made. When correct diaphragmatic breathing is restored by re-education, these symptoms disappear, and no patient should be discharged before it has been obtained. Failure to re-educate the diaphragm after prolonged rest is a common cause of the illness of the patient who has "never been well since the operation." In certain obstinate cases where puncture has to be made to restore activity, carbon dioxide inhalations might quite well be tried first. Such cases are very rare.

I have endeavoured to stress the importance of deflation as occurring after operations; its recognition is equally necessary in the consideration of many other pulmonary conditions. Though frequently preached, the significance of deflation has not yet been generally assimilated.

Conclusions.

(1) The total volume of the chest is diminished when the reclining or supine posture is assumed. (2) The capacity and expansion of the upper part of the chest are increased. (3) The capacity and expansion of the lower part are diminished. (4) Inflation of the upper lobe depends on expansion of the upper part of the thorax, combined with tone in the diaphragm. (5) Inflation of the lower lobe is dependent on a low level of the diaphragm, and probably on inspiratory descent of the costal portion. (6) Deflation occurs in all persons who are quiescent when reclining or supine, and therefore is present in most of our bed patients, particularly those subjected to operations. (7) In the great majority of cases no symptoms follow. (8) Infection, if present beforehand, may be spread. Frequently it is acquired at the time of the operation. (9) Therapeutic expansion of the lung in the early days after operation as a routine measure is not advisable; but (10) Before discharge, voluntary control over the diaphragm should be fully acquired,

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ALBUMINURIA COMPLICATING PREGNANCY.

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PART I.*—THE DANGERS.

So much has been heard lately of the maternal risks that pregnancy, especially abnormal pregnancy, entails that it will not be out of place to consider the ultimate reason for taking these risks. The reason, of course, is the child; and the greater the value placed upon the child, the greater will be the maximum risk that is considered justifiable for the mother. In the particular instances that we are considering—i.e., pregnancies complicated by albuminuria—it will be an advantage to know exactly how often a fruitful result is attained. There can be no justification, beyond the hope that it might have been different, in taking any avoidable maternal risk for the sake of an abortion, a macerated fœtus, or an infant which dies in early infancy from prematurity.

In cases of albuminuria the dangers to the fœtus are two: (1) the risk of abortion or death in utero as the result of the toxæmia; (2) the risk of death from prematurity in early infancy in cases where pregnancy has been terminated either accidentally or by intent, sometime before full term. In the

* Part II., dealing with treatment, will appear next week.

following Table the results to the fœtus of pregnancies complicated by albuminuria are shown. Abortion, macerated fœtus, and death in early infancy from prematurity only are mentioned, since deaths due to accidents of labour, and infant deaths due to causes other than prematurity, cannot reasonably be assigned to the toxæmia in the mother. The cases have been grouped according to the renal condition of the mother during pregnancy.

TABLE I.

Type of renal lesion in mother.	Pregnancies.	Result to fœtus. Abortion.	Macerated fœtus.	Death from prematurity.	Total deaths attributable to toxæmia per cent.
Chronic nephritis	20	8	2	2	60
? Chronic nephritis	63	14	18	2	54
Not chronic nephritis ..	141	1	12	4	12

The figures in Table I. under the heading of abortion are certainly relatively too low, since abortions do not as a rule come under the care of the maternity department and, especially in the chronic nephritis group, the number of pregnancies which end in abortion is much greater than these figures suggest. This criticism does not, however, apply to the patients in whom no chronic renal lesion was present, since in this group the toxæmia is seldom present before the fœtus is viable.

Considering the total fœtal death-rate, we see at once that when pregnancy occurs in a woman already suffering from a chronic renal lesion the chance of a fœtal catastrophe is very great, so that in allowing pregnancy to proceed in such a patient we are allowing the mother to take a considerable risk for the sake of a very problematical infant.

In a toxæmia of pregnancy that is not superimposed upon a chronic renal lesion, however, the chance of a living child is tolerably good (i.e., only 12 per cent. below the chance in a normal pregnancy), and whatever risk we may be allowing such a mother to take, we have at least the justification of knowing that it is taken probably for a living child, and not merely for an abortion or macerated fœtus.

The patients from whom these deductions have been drawn were all under the care of the Guy's Hospital Maternity Department, where induction of premature labour for toxæmias of pregnancy is resorted to more frequently and earlier than in many other institutions; yet in spite of this more infant life was lost from maceration in utero than from death from prematurity in early infancy. These two dangers are reciprocal to some extent so far as radical prophylaxis is concerned. A diminution in the death-rate from prematurity could be obtained at the expense of an increase in the death-rate from maceration in utero by delaying labour; but the figures quoted show that in the present state of practice maceration in utero is still a greater danger than death in early infancy from prematurity.

Dangers to the Mother.

(1) DANGER OF ECLAMPSIA.

Eclampsia is such a well-recognised and such a dreaded complication of albuminuria occurring during pregnancy that it would seem to be unnecessary to stress its danger. In spite of this, however, it appears that the onset of eclampsia is still sometimes looked upon as merely bad luck, something entirely outside our control. The recent report of the Maternal Mortality Committee [1] showed that out of 218 women dying from eclampsia albuminuria had been known to exist previously in 62, but in only 21 of these women was any attempt made to give reasonable treatment for the albuminuria before the onset of eclampsia. A fatalistic outlook towards the onset of eclampsia is absolutely unjustified, as there is ample evidence that this disease can be almost entirely prevented by early detection, and by early and wholehearted treatment of albuminuria.

During a period of five years about 8000 women domiciled in the Guy's Hospital maternity district were confined under the care of the hospital (Class A patients in Guy's Hospital Maternity Report); amongst these there were about 300 cases of albuminuria during pregnancy. There were two cases of eclampsia: the first occurred in a patient who was under treatment for albuminuria and who refused induction of premature labour two weeks before the onset of fits. The second was a patient who had been examined a fortnight before the onset of fits, and in whom the urine was then free from albumin. Thus, excluding the patient who refused treatment, the only case of eclampsia that occurred was a fulminating case. No case occurred in a patient actually under treatment for albuminuria. This is an important fact since, provided adequate treatment of albuminuria is forthcoming, the danger of eclampsia lies not so much amongst those people whom we know to have albuminuria, as amongst those whom perhaps a fortnight previously we knew to be albumin-free. Fulminating eclampsia will still remain with us, since the very suddenness of the toxæmia precludes the possibility of giving adequate treatment to the preceding albuminuria.

This is, perhaps, the most favourable thing that can be said about albuminuria of pregnancy—viz., that provided adequate treatment is given eclampsia can be avoided. The care entailed in the term "adequate treatment" is exacting and tedious, but it is so important that it will be considered separately in a later section.

(2) DANGER OF PATENT CHRONIC NEPHRITIS.

Until recently it was generally assumed that, provided eclampsia could be avoided, the bad sequelæ of the toxæmias of pregnancy were of practically no importance, on account of their supposed rarity, and furthermore that the history of one attack of albuminuria did not in any way affect the prognosis of a subsequent pregnancy. In spite of the fact that this belief was so widely held, when the evidence for it was sought it was found to be based upon a pious hope rather than upon any accurate investigation.

The fact that clinical experience in cases of toxæmia of pregnancy pointed to exactly opposite views, led me in 1927 to investigate the late effects in 78 cases of albuminuria of pregnancy occurring in the practice of Guy's Hospital,[2] and the results of this study showed, amongst other things, that out of 37 previously healthy women who suffered from albuminuria of pregnancy five (14 per cent.) had undoubted signs of chronic nephritis when examined six months to two years after delivery. Furthermore, excluding patients in whom there was any suggestion of a pre-existing chronic nephritis, it was shown that out of 21 patients who became pregnant again after one attack of toxæmia, 12 had a recurrent albuminuria—a recurrence rate of 57 per cent. If these

figures are a true index of the position, it is clear that the late sequelæ of the toxæmias of pregnancy assume a very great importance, and though less dramatic than the immediate dangers (eclampsia and death of the fœtus in utero) their importance lies at least in their comparative frequency.

In support of the figures obtained at Guy's Hospital I am able to quote those of James Young,[3] who found that the incidence of chronic nephritis following eclampsia was 3 per cent. and following albuminuria 8 per cent. The same observer [4] has found that the recurrence-rate for albuminuria is 31 per cent. Caldwell and Lyle [5] found that chronic nephritis resulted in 8 per cent. of eclamptic patients. Gibson [6] found the incidence of chronic nephritis following eclampsia to be 35 per cent., and following albuminuria in previously healthy women, 16 per cent. He also quotes Koblanck, who found that 6·5 per cent. of patients developed chronic nephritis as a result of a toxæmia of pregnancy. Harris [7] found that eclampsia was followed by chronic nephritis in 11 per cent. of cases, and the "pre-eclamptic toxæmia" yielded 60 per cent. of chronic nephritis. Kebes [8] found that 25 per cent. of eclamptic patients and 60 per cent. of "pre-eclamptic" patients had signs of chronic nephritis when examined at long intervals after delivery. Kellog [9] states that toxæmia of pregnancy recurs with a subsequent pregnancy in 15 per cent. of cases.

It is impossible to compile a parallel array of figures in support of the old idea that albuminuria of pregnancy does not recur or give rise to chronic nephritis, since a search through the literature has revealed nothing beyond mere expressions of opinion based upon no accurate observation, and repeated with the monotonous regularity of an article of faith.

No one weighing all the available evidence fairly can possibly escape the conclusion that the old view is wrong; but old beliefs die hard, and for this reason it has been thought worth while to extend the investigation into the late results of albuminuria, and to add to the existing evidence that chronic nephritis is an important sequel of pregnancy toxæmia, and that albuminuria of pregnancy frequently recurs with subsequent pregnancies.

The present investigation is concerned with 45 patients with albuminuria complicating pregnancy who have been examined at intervals of from one to two years after delivery, but to this number have been added the 44 cases which formed the basis of a similar investigation the results of which were published in 1928.[2] Thus the complete series consists of 89 patients. They are classified according to the presumed state of the kidneys before the pregnancy during which the albuminuria occurred. This has not always been an easy task, since it is difficult in many cases to give a positive statement as to the entire absence of chronic renal damage. For this reason a class has been kept separate for those patients in whom there was any doubt as to whether or not the renal function was normal before pregnancy.

There are thus three classes to be considered, the classification depending upon the condition of the kidney before the advent of the pregnancy in which the albuminuria occurred.

1. *Chronic Nephritis.*—All the patients in this group had signs, either before pregnancy or during the early months of pregnancy, which suggested permanently damaged kidneys.

2. *? Chronic Nephritis.*—In the majority of patients in this group there was probably no permanent renal damage, but either on account of some suggestive history (such as repeated sore-throats, or œdema, or headaches), or because of the absence of positive evidence that the urine was albumin-free before or at the beginning of pregnancy, they have been put in this doubtful group to ensure that the third group may be free from any doubtful cases.

3. *Not Chronic Nephritis.*—The cases included here gave no history suggestive of previous renal disease, had normal cardiovascular systems, and in every case the urine was albumin-free when first examined, either before or at the beginning of pregnancy. If there was any doubt at all, either on consideration of history or physical signs, the patient was placed in the previous group.

All these patients were again examined at intervals varying from one to two years after delivery, and they were re-grouped according to the renal condition as it then existed. The state of the kidney was determined on this occasion by a consideration of history, symptoms, and physical signs, including an examination of the cardiovascular system, fundus oculi, and urine, and in certain cases this was supplemented by biochemical tests. The results are given in Table II.

TABLE II.

Groups—I. C.N. before pregnancy. [1] 3 cases.			II. ? C.N. before pregnancy. [2] 7 cases.			III. Not C.N. before pregnancy. [42] 79 cases.		
State of kidney after delivery.								
C.N.	? C.N.	Not C.N.	C.N.	? C.N.	Not C.N.	C.N.	? C.N.	Not C.N.
[1] 3	—	—	[2] 5	1	1	[4] 9	[4] 11	[34] 59

C.N. = Chronic nephritis.

The figures in brackets refer to cases in the present investigation, and the other figures are obtained by adding these to the corresponding figures of a previous series which was published in 1928.

Since all the patients in Group I. were considered to have a definite chronic nephritis before the pregnancy in which the albuminuria occurred, all of course showed signs of chronic renal disease when examined after delivery, and in many cases at least it was apparent that the amount of renal damage had increased as a result of the pregnancy. It is, however, impossible to assess definite relative values to different degrees of chronic nephritis, so that no very valuable conclusions can be drawn from the results in this group.

Group II. consisted of patients in whom the diagnosis before pregnancy was doubtful, and for this reason no attempt has been made to draw conclusions from it. It may be significant, however, that when examined after the attack of albuminuria there were five cases in which a diagnosis of definite chronic nephritis was made.

All the patients in Group III. may fairly be assumed to have been healthy women before the pregnancy toxæmia occurred; and, although the difficulties in making a certain negative diagnosis are very great, yet the inclusion in Group II. of all doubtful cases has made it possible to rely upon Group III. to contain only initially healthy women. It is seen, therefore, that out of 79 healthy women who had albuminuria of pregnancy 9 (11·4 per cent.) developed definite chronic nephritis immediately following upon the toxæmia and, in addition to this number, in 11 (14 per cent.) it was not possible to be sure of the kidney condition after delivery. Thus in only 59 (75 per cent.) could it be said at all definitely that the kidney was not permanently damaged when the patient was subjected to a routine examination one to two years later.

(3) DANGER OF RECURRENT ALBUMINURIA (OCCULT NEPHRITIS).

In 52 patients it was possible to observe the course of a pregnancy subsequent to the one in which albuminuria first appeared, and to note whether or not a recurrent albuminuria occurred. The results of these observations are shown in the following Table. All patients had had an attack of albuminuria in a pregnancy previous to the one under consideration, and Table III. shows the proportion of these patients in which the albuminuria recurred with the subsequent pregnancy.

TABLE III.—*Result of Pregnancy following the First Pregnancy with Albuminuria.*

Groups—I. C.N. before first pregnancy with alb. [1] 2 cases.		II. (?) C.N. before first pregnancy with alb. [3] 9 cases.		III. Not C.N. before pregnancy with alb. [20] 41 cases.	
Recur. alb.	No recur. alb.	Recur. alb.	No recur. alb.	Recur. alb.	No recur. alb.
[1] 2	—	[3] 9	—	[12] 24	[8] 17

Recur. alb. = recurrence of albuminuria.

The figures in brackets refer to cases in the present investigation, and the other figures are obtained by adding these to the corresponding figures of the previously published series.

It is obvious that all patients with chronic nephritis (Group I.) will have recurrent albuminuria. Group II., consisting as it does of doubtful cases, is of little value for the purpose of drawing conclusions; but in Group III. we see that out of 41 initially healthy women who apparently recovered their normal kidney function completely after one attack of albuminuria and who subsequently became pregnant again, 24 (59 per cent.) suffered from a recurrent albuminuria.

Significance of Recurrent Albuminuria.

The significance of recurrent albuminuria is open to several mutually antagonistic interpretations, but three in particular must be considered.

In the first place it had been widely assumed that the mere fact of recurrence was proof that a chronic nephritis existed before the onset of the first albuminuria. This opinion was based merely upon a slavish belief that toxæmia of pregnancy did not recur, nor give rise to chronic nephritis, and therefore that an albuminuria that did recur must of necessity be associated with a pre-existing chronic nephritis, and by definition could not possibly be a simple toxæmia of pregnancy. There never was any evidence in favour of this opinion, and a sufficiently large number of cases have now been analysed to make it certain that a large proportion (probably 50 per cent.) of initially healthy women who have suffered from albuminuria with one pregnancy do have recurrence with the next, and that a smaller proportion (probably 10 per cent.) develop an obvious chronic nephritis as an immediate result of their first toxæmic pregnancy. The recognition of this now established clinical fact is all that is necessary to explode for ever the idea that recurrent albuminuria is ipso facto proof that a chronic renal lesion preceded the first pregnancy with albuminuria.

Two other interpretations of the significance of recurrent albuminuria with apparently normal renal function in the interval between pregnancies have been put forward. In 1927 James Young [4] suggested that those women who develop albuminuria with pregnancy have some inherent predisposition to toxæmia of pregnancy; that this "unknown factor" stays with them even though their kidneys recover completely after delivery, but that it determines the onset of another attack of albuminuria with each successive pregnancy. In other words, he regards the kidneys as really (not merely "apparently") healthy in the intervals between pregnancies, the patient as abnormal only when she is pregnant, and the cause of her abnormality an inherent and unknown rather than an acquired factor. This view has the advantage that it explains why such patients are free from the stigmata of renal disease in the intervals between pregnancies, but the assumption of an unknown, inborn, specific idiosyncrasy to toxæmia of pregnancy is undesirable because there is no direct evidence of its existence, and because it does not clarify the situation to replace one enigma by another nor does it guide us in our search for improved methods of treatment to assume an "unknown inborn" cause which we cannot control.

In 1928 [2] I first suggested that the facts associated with recurrent albuminuria could best be explained by assuming that these patients had, as a result of their first toxæmia pregnancy, suffered some permanent renal damage which, although insufficient to give rise to signs of chronic nephritis, was yet sufficient to give rise to albuminuria when the strain of a subsequent pregnancy was imposed. In other words, recurrent albuminuria is merely a result of a subliminal pregnancy toxæmia acting in a patient with a low-grade chronic nephritis. The result of further investigations served to strengthen my belief, and in 1929 [10] I first suggested the term "occult nephritis" to denote the condition of the kidney in these patients. The lesion is hidden except when the most delicate test of renal function is applied—the test of pregnancy. In support of this view may be used its reasonableness, since all the known facts can be explained without invoking any new unknown factor.

It is generally accepted that albuminuria (apart from that occurring during labour) occurs in from 3 to 5 per cent. of previously healthy pregnant women—i.e., this proportion of patients develop a toxæmia of sufficient severity to cause obvious signs, but apart from these the remaining 95 per cent. probably all have a toxæmia of lesser severity which is so slight as to pass undetected provided that the renal function is not in any way subnormal before the pregnancy. In a patient with slight renal damage (occult nephritis), however, the very slight toxæmia associated with any pregnancy will probably be sufficient to cause an albuminuria every time, although in the intervals between pregnancies all stigmata of renal disease may be absent. If we inquire into the original cause of this occult nephritis we have only to remind ourselves that 10 per cent. of previously healthy women (as the result of a first attack of albuminuria of pregnancy) develop patent chronic nephritis, so that it is perfectly reasonable to find that a larger proportion (50 per cent.) develop occult nephritis. The point which cannot be stressed too strongly is that the foundations for recurrent toxæmia (occult nephritis) are laid during the first toxæmia that the patient experiences.

There are two simple observations which lend support to the theory that the tendency to recurrent albuminuria is an acquired disability rather than an inborn idiosyncrasy. In the first place, I have shown that out of 57 patients in whom a toxæmia of pregnancy occurred for the first time 40 per cent. were multiparæ—i.e., these 40 per cent. at least had no inborn predisposition to albuminuria,[2] since they had previously gone through one or more pregnancies

with no signs of renal disorder. In the second place, I have shown[10] that the incidence of recurrent albuminuria amongst initially healthy women can be diminished by early induction of labour during the first toxæmic pregnancy, and this is very strong evidence that the first toxæmia, if at all prolonged, may cause an occult nephritis, which is the basis for all recurrence of albuminuria with subsequent pregnancies.

The earlier observations on the influence of the duration of the first toxæmia on the subsequent recurrence-rate have been extended recently, and the following analysis of patients is obtained by adding 20 new cases to the 47 similar cases which were published in a previous paper. Thus 67 initially healthy patients were observed during at least two pregnancies, the first of which was associated with albuminuria. As the result of their first toxæmia 10 of these developed signs suggestive of permanent renal damage, thus leaving 57 in whom there were no signs of chronic nephritis. All these 57 patients became pregnant again, and they have been divided into two groups: (1) those in whom the first toxæmia lasted less than three weeks; (2) those in whom the first toxæmia lasted more than three weeks. The recurrence-rate for albuminuria with the next pregnancy has then been worked out for each group. (Table IV.).

TABLE IV.

First toxæmic pregnancy.	First toxæmic pregnancy.					
	Lasted less than 3 weeks. [10] 38 cases.			Lasted more than 3 weeks [10] 29 cases.		
Non-pregnant interval.	C.N.	No alb.		C.N.	No alb.	
	[1] 2	[9] 36		[3] 8	[7] 21	
Subsequent pregnancy.	—	Result of next pregnancy.		—	Result of next pregnancy.	
		Alb. [2] 13	No alb. [7] 23		Alb. [6] 16	No alb. [1] 5

The figures in brackets refer to new cases that have been investigated: the other figures are obtained by adding these to the corresponding figures of the series published previously.

These results show quite clearly that amongst 38 previously healthy patients in whom pregnancy was terminated within three weeks from the time that the toxæmia was first noted, only 5 per cent. developed signs of permanent renal damage. Amongst 29 patients in whom pregnancy was allowed to continue longer than three weeks after the toxæmia was first noted, 28 per cent. developed signs of chronic nephritis. This suggests that the longer the first toxæmia is allowed to continue the greater is the danger of permanent renal damage.

All the patients in whom there were no obvious signs of permanent renal damage after the pregnancy subsequently became pregnant again, and the Table shows clearly that the recurrence-rate for albuminuria was 36 per cent. amongst those whose first toxæmia lasted less than three weeks, and 76 per cent. amongst those whose first toxæmia lasted more than three weeks. This suggests that the longer the first toxæmia is allowed to continue the greater is the danger of a recurrence of albuminuria with a subsequent pregnancy, even though there may be no obvious signs of chronic nephritis in the interval between the pregnancies. The foundations for the recurrent albuminuria are laid down during the first toxæmia. The foundations are, in fact, an occult nephritis.

Difficulties in the Theory of Occult Nephritis.

One difficulty in accepting this theory is that it makes pregnancy the most delicate test of renal function that we possess. To those of us who have been disappointed at the practical results of biochemistry in elucidating the problems of the toxæmias of pregnancy this comes as no surprise, and there is no real difficulty in assuming that a renal lesion may exist without any obvious alterations in the blood chemistry. In fact the extensive work of Hewitt, Couper, and Cruickshank[11] has established the striking absence of marked biochemical changes in the majority of the toxæmias. Furthermore, there is nothing fantastic in the belief that a chronic nephritis may exist without any of the usually accepted clinical signs—e.g., albuminuria. Albuminuria may be a sign of active progressive renal disease, but its absence tells us nothing about an old healed renal lesion. In this connexion F. J. Browne[12] has recently supplied experimental evidence showing that in rabbits known to have chronic renal damage there may be no albuminuria in the intervals between pregnancies, and yet albuminuria may occur during the pregnant state. He has thus supplied experimental proof of the existence of the condition which I have termed occult nephritis, and in his rabbits pregnancy was the most delicate clinical test of renal function that he could apply.

The other difficulty in accepting the theory of occult nephritis is that, although as a general rule a patient with this condition has albuminuria with every pregnancy, exceptions sometimes occur. It is possible for a woman to have two or three or even more consecutive pregnancies associated with albuminuria, and yet have a subsequent one in which no albuminuria occurs. Furthermore, an albuminuria starting, say, at the thirtieth week with one pregnancy, may not occur in the next until much nearer full term. The conception of occult nephritis that has been outlined might seem to require that if albuminuria occurred with two successive pregnancies it should invariably recur with all, and furthermore that it should tend to occur earlier and earlier with each successive pregnancy. This is in fact what usually happens, but there is no need to abandon the theory for the exceptional cases, since it must not be forgotten that recurrent albuminuria is to be looked upon as the result of two factors—an occult nephritis which is permanent plus a toxæmia which is temporary and the result of the particular pregnancy under consideration. Generally speaking, the result of these two factors is a clinical albuminuria, but exceptionally the factor supplied by the pregnancy—the toxæmic factor—may be so small as not to give rise to albuminuria even in a patient with occult nephritis. Thus we can explain the exceptional cases, where an apparently normal pregnancy may follow several in which albuminuria has occurred, without abandoning our views on the permanence of the chronic renal lesion, which in these cases is "occult" even during pregnancy. A similar explanation can be supplied to those cases which do not show an invariable increase in severity of symptoms with successive pregnancies. Here again F. J. Browne has given us the experimental evidence for this conception that has hitherto been lacking; he has found precisely these exceptional clinical manifestations in rabbits which were known to have occult nephritis.

Summary.

From the foregoing considerations it is clear that a previously healthy patient suffering from albu-

minuria during pregnancy is running several risks, which may conveniently be summarised as follows:—

1. *The danger of eclampsia.*—We have seen that, provided reasonable care and treatment are forthcoming, this is very small.

2. *The danger of developing patent chronic nephritis.*—With the standards of treatment that have in the past been considered adequate this danger is considerable—about 10 per cent.

3. *The danger of developing occult nephritis.*—With the standards of treatment that have in the past been considered adequate this danger is probably in the region of 50 per cent.

4. *The danger of losing her fœtus by maceration in utero.*—When induction of premature labour is resorted to for inductions that have in the past been deemed reasonable, this danger is about 9 per cent.

5. *The danger of losing her infant in the neonatal period from prematurity.*—When induction of premature labour is resorted to for inductions that have in the past been deemed reasonable this danger is about 3 per cent.

The above estimates of the risk are true only for initially healthy patients. If there is any permanent renal damage before pregnancy the risk becomes all the greater, but even as it is set out above it is surely sufficient cause for us to look critically upon our accepted treatment of toxæmia of pregnancy, in the hope that we may find some places at least where improved methods may be instituted.

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THE ÆTIOLOGICAL IMPORTANCE OF THE ELEMENTARY BODIES IN VACCINIA AND FOWL-POX.

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THE purpose of this brief communication is to place on record some new observations which, so far as they go, support the claim that the Paschen and Borrel bodies met with in the lesions of vaccinia and fowl-pox represent the actual viruses of these respective diseases. During the past six years, while occupied with various studies on these viruses, I have given a good deal of attention to these minute bodies, which occur with absolute constancy in the lesions, and are readily detectable by appropriate staining methods. That in the early stages of the vaccinial lesion in the skin of the rabbit these bodies increase enormously in numbers and appear to be quite free, I have noted elsewhere. In the later or healing stages of the lesion the bodies congregate in the neighbourhood of the inflammatory cells of the exudate, and the impression obtained has been that the bodies are actually phagocyted, though, so far, phagocytosis has not been proved, owing to technical difficulties of demonstration, which, however, may yet be overcome. The behaviour of the bodies from their first appearance in the lesion, their increase in numbers as the lesion progressed, and their final aggregation among cells in the exudate suggested analogies at least with that of frankly bacterial invaders associated with skin phlegmons.

In fowl-pox the status of the Borrel bodies as possible agents of this disease has very materially advanced in the past three years, particularly since the experimental demonstration by Woodruff and Goodpasture (1929) that a single Bollinger inclusion body can infect. As fowl-pox virus is filtrable through the lower grade filters which retain bacteria, the infecting agent in the large Bollinger body must necessarily be something considerably smaller. Since then I have, like the American workers, been successful in demonstrating that the large Bollinger inclusion body which is so striking a feature of the fowl-pox lesion is really an aggregation of the minute Borrel bodies. Giemsa-stained preparations showing this relationship between the Bollinger and Borrel bodies were demonstrated at the International Congress of Botany in Cambridge in August, 1930, and at the Pathological Society of Great Britain and Ireland in January, 1931. To many minds this proved relationship between a minute body of dubious nature and a large structure like the Bollinger body, which has been known and variously interpreted for 60 years, would, of itself, supply cogent evidence for the view that the Borrel body represents the actual virus. I may add here that in potent bacteria-free filtrates, both of fowl-pox and of vaccinia virus, I have succeeded in demonstrating elementary bodies in stained deposits from filtrates spun at high speed. It was obvious, however, that other lines of inquiry which might yield crucial evidence in support of the ætiological relationship had to be explored. One line of inquiry was clearly serological, in view of the mass of evidence now available that extracts prepared from vaccinial and small-pox lesions fix complement, or are specifically precipitated, in the presence of anti-vaccinial sera. The work of Gordon (1925), Bedson and Bland (1929), Burgess, Craigie, and Tulloch (1929), and Craigie and Tulloch (1931) may be alluded to in this connexion.

The only evidence I can find bearing on the influence of specific immune serum on these elementary bodies is that recorded by Prof. Paschen, who found, and figured, in stained preparations of precipitates obtained by the interaction of "immune serum" and lymph from vaccinated children, agglutinated masses of elementary bodies.

During the past five years I have made repeated but, until recently, quite unsuccessful attempts to secure from vaccinial and fowl-pox lesions pure suspensions of elementary bodies that might be employed for serological test, and especially for agglutination. From the repeated failures it gradually became clear that certain requisites were essential for success. It was advantageous to employ material from early lesions (on skin of rabbit or comb of fowl) in which the elementary bodies were numerous and free. Such material was triturated thoroughly in a mortar with distilled water. The crude suspension was then shaken up in a flask with at least an equal volume of ether, and allowed to stand on the bench for at least 24 hours, being frequently shaken up

during this period. The suspension was then centrifuged at high speed. The upper layer of ether and the large middle layer of light fatty matter which had risen to the surface were removed, leaving an underlying opalescent fluid, and occasionally a small deposit.

This underlying layer, together with the deposit, was again centrifuged at high speed for at least one hour (9000 revolutions per minute). The resulting deposit was collected after discarding the supernatant fluid. This small deposit, consisting of elementary bodies and some debris of cells, was then taken up and suspended in formolised saline (1 in 400). By further fractional centrifugalisation of this suspension and natural sedimentation, it was possible to eliminate practically all of the grosser debris, leaving only the elementary bodies. At all stages of the process the degree of refinement of the suspension was controlled by microscopical examination of appropriately stained samples.

Microscopical examination of the final suspensions in formolised saline by Giemsa (long method) or carbol fuchsin, preceded by Loeffler's flagella mordant, revealed a carpet of uniformly distributed elementary bodies.

AGGLUTINATION TESTS.

These suspensions have been tested with various sera but, so far, only on a small scale. The sera include normal fowl and rabbit sera, fowl sera, both from recovered fowl-pox and from hyper-immunised fowls, and antivaccinial sera, obtained by hyper-immunisation of rabbit and horse. The tests have been most conveniently made in hanging drops on coverslips in vaselinised chambers, which permit the microscopical examination of the progress of any reaction taking place between the two reagents. It has emerged that the elementary bodies of fowl-pox are not clumped by sera of young normal chicks in dilutions of 1 in 5, but are clumped by the sera of fowls at various periods after recovery from fowl-pox in dilutions ranging from 1 in 20 to 1 in 160. Sera of hyperimmunised fowls also clump the elementary bodies strongly in fairly high dilution. Similarly the elementary bodies of vaccinia (Paschen bodies) are clumped by hyperimmune sera in high dilution, but not by normal rabbit sera in dilution of 1 in 5. The agglutination experiments so far have been largely qualitative, with a view to establishing the fact that the elementary bodies are agglutinated in the presence of the corresponding antiviral serum. Quantitative titrations will be undertaken later, now that a satisfactory technique has been secured.

The clumping of the elementary bodies in the hanging drops can be readily followed, and the picture presented is in no way peculiar. The clumps have also been examined by various staining methods, which clearly reveal their composition.

SUMMARY AND CONCLUSIONS.

Homogeneous suspensions in formolised saline of the elementary bodies met with in vaccinia and fowl-pox (Paschen, 1906; Borrel, 1904) have been secured, which are free from other elements. The bodies in such suspensions are agglutinated by the sera of animals (fowls and rabbits) which have recovered from fowl-pox or vaccinia respectively or which have been hyperimmunised. So far as tests have revealed, they do not appear to be agglutinated, even in dilutions of 1 in 5, by normal sera. The agglutinated masses from hanging drop experiments have been appropriately stained, and shown to consist solely of these bodies.

The new data support the original claims of their discoverers, on grounds of association alone, that the elementary bodies represent the actual infecting agents. With recently reported improvements in methods for cultivating certain viruses in vitro it may be possible to determine whether growth of "virus" as determined by animal experiment is associated with corresponding growth of elementary bodies.

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INFANTILE HYPERTROPHIC PYLORIC STENOSIS.

SYMPTOMATOLOGY, TREATMENT, AND PROGNOSIS.

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ALTHOUGH infantile hypertrophic pyloric stenosis is observed relatively infrequently in a general hospital, it has seemed profitable to study a small series of patients with special reference to the after-history in relation to the treatment adopted. At the Middlesex Hospital, between the years 1919 and 1930, 18 children were treated for this malady. I have personally observed and followed up seven of these, and I have also analysed the remaining 11 cases and investigated their after-histories.

If the 18 cases are arranged chronologically it is found that the first five (1919–1924) were treated medically, while the remaining 13 were treated surgically by Rammstedt's operation. This illustrates the trend of opinion in this country. In Barrington Ward's series [1] of 359 cases, operative treatment was adopted in 310. In 1917, one case was operated upon and died; of nine cases treated medically seven died. In 1925, 54 cases were operated upon with one death (1·8 per cent.); one case was treated medically and died. In 1927, 25 cases were operated upon, only one child dying (4 per cent. mortality); no case was treated medically. In Poynton's [2] series of 100 cases there were 20 deaths, a mortality of 20 per cent. In the present series of 18 cases, all five patients treated medically recovered, while among the remaining 13 treated by Rammstedt's operation there were three deaths.

Wolff,[3] of Eisenach, gives a series of 98 cases from 1912 to 1930. Of these only six were operated upon, and among the 92 treated medically there were only two deaths. These are truly remarkable figures and form an interesting contrast with the method of treatment and results obtained in this country.

Ætiology.

In three patients the symptoms began at or soon after birth. In the remaining 15 the average age of onset was 3½ weeks, the earliest being 12 days and the latest 5½ weeks. There was the usual preponderance of males, all but one being of this sex. G. F. Still drew attention to the frequency with which first children are affected. In 299 cases in Barrington Ward's series, 171 (57 per cent.) were first children of

the family. In the present series of 17 male children, eight (47 per cent.) were in this category.

Although *phimosis* was at one time considered to play a part in the production of the condition, it is difficult to understand its influence. Of 17 patients, four were Jewish and circumcision had been performed on the eighth day. In two other cases circumcision had been performed on the ninth and eleventh day respectively. Amongst the remaining 11 cases, phimosis was said to be present in four only. In one of these, circumcision and medical treatment led to a complete cure without operation. In another, circumcision was performed at the same time as the Rammstedt operation. Of 12 patients still living, five are uncircumcised. These findings suggest that phimosis was not an ætiological factor.

In none of the cases was there any relationship between the parents. All except two of the children were breast-fed from birth for varying periods before other preparations were tried. One of these two was fed on Allenbury's Food No. 1 up to the time of onset of symptoms. The other was fed on Nestlé's Milk, as "he did not take to the breast."

Symptoms.

Of the symptoms, vomiting was the most important. In 14 cases it was forcible or projectile in character. It usually occurred immediately after each feed, but occasionally after every third, fourth, or fifth feed, and rarely were all feeds vomited together at the end of the day.

The forcible nature of the vomiting was very characteristic, and was described variously as being projected from 2 ft. to 3 ft. "right over the mother's clothes" or "over the side of the cot." In other cases it was described as being "just pumped up all at once," "whole of feed returned through mouth and nose as if pumped up," and "spout out as from a water tap." In three cases no note was made as to the character of the vomiting, and in one case it was not projectile and "just ran out of his mouth" at first once a day, then twice or three times, and then almost after every feed.

In three cases there had been slight vomiting after food from birth. In two of these it was said to be projectile in character.

Constipation was present in 14 cases and became marked when vomiting commenced, or shortly afterwards. Rarely constipation preceded the vomiting. In four cases it was absent, the bowels acting regularly or freely after each feed. Green stools were present in seven cases.

Wasting was present and usually marked in all cases. In 14 children in whom the birth-weight is known nine on admission were under the birth-weight. This drop in weight varied from 1 oz. to 1 lb. 10 oz., with an average of 10 oz.

Jaundice was present in one case for one week before admission, but had almost disappeared at the time of operation. The Wassermann reaction was negative.

Physical Signs.

A *pyloric tumour* was palpated in 11 of the 18 cases. In four patients no note was made as to whether a tumour was present or absent. All four were operated upon and a tumour found. In three cases palpation of the abdomen failed to detect any tumour. Only one was operated upon and a definite tumour was found at the pylorus.

In these patients, where a tumour was detected, it was usually found just above and to the right of the umbilicus. Occasionally it will be found rather far to the right, almost in the right lumbar region. The position of the tumour is not constant and may vary in place from time to time as a wave of peristalsis passes over it. As this wave reaches the pylorus it may be felt to contract and harden. In one case it was noted that the liver reached a distance of three fingers' breadth below the costal margin; this relative enlargement of the liver is common and partly accounts for the difficulty in detecting the tumour.

Visible gastric peristalsis was a prominent sign in 14 cases. In the remaining four it was, in all probability, also present, although no note was made to this effect. The abdomen is uncovered and watched during a demonstration feed. The wave appears as a large rounded ball near the left costal margin, passes across the epigastrium from left to right, and ends as a smaller rounded ball, as a rule just above and to the right of the umbilicus, but depending on the position of the pylorus. The rate is usually fairly rapid.

Medical Treatment.

This commenced with a glycerin enema ℥ iii, which was usually repeated in four to five days and then as often as necessary. Gastric lavage with a sodium bicarbonate solution ℥ i in a pint of boiled water was employed at first either twice or three times a day, reducing later to once a day, and finally omitting altogether about five to six weeks after commencing treatment.

Method of feeding.—One child was breast-fed throughout with supplementary feeds of citrated milk at night. Thirteen days after admission a phimosis was dealt with by "slitting up" of the prepuce, and vomiting occurred on only three occasions afterwards, and in three weeks the child had gained 5 oz. There was a final gain in weight of 14 oz. during a stay of seven weeks. Artificial feeding was employed in the other four children, e.g., as follows:—

Peptonised milk ℥ iss and boiled water ℥ss were given two-hourly by means of a stomach-tube. As an alternative albumin water ℥ii may be given. These were replaced subsequently by the same quantities of citrated milk and boiled water given at first by the stomach-tube, then by spoon, and finally by bottle. The amount was gradually increased until citrated milk ℥v was being given three-hourly.

Drugs.—In one case tinct. opii ♏ i with haustus bismuthi ℥ i was given six-hourly for diarrhœa with green stools. In another the following drugs were employed: tinct. belladonn. ♏ iii, tinct. camph. co. ♏ ii, and sodium bicarb. grs. iii. In all five children the vomiting gradually diminished, and finally ceased altogether. In one child visible gastric peristalsis continued, and had not completely disappeared on discharge from hospital. This child gained 5 lb. 8 oz. in five months. The other children gained 2 lb. 8 oz. in three months, 1 lb. in five weeks, and 14 oz. in two months, respectively.

Although constipation was a marked feature in all, the bowels became regular during treatment, two to three motions occurring every day.

Surgical Treatment.

PRE-OPERATIVE.

Although operation is undertaken as soon as possible after the diagnosis is made, at least 24 hours are required to prepare the child. It is usually performed on the first, second, or third day after admission. One child succumbed in which operation was delayed for 32 days after admission.

Twenty-four hours before operation enemata 3 oz. of normal saline with 5 per cent. glucose are commenced and repeated six-hourly. The child is breast-fed, if possible, every three hours until four hours before the time of operation, after which nothing further is given by the mouth. The last two feeds are given with a teaspoon. Subcutaneous saline 1½ oz. to 3 oz. with 5 per cent. glucose, is given into each axillary region 12 hours before and again two hours before operation into the thighs. Finally, 1½ hours before operation, the stomach is washed out with plain water, and the limbs are wrapped in warm cotton-wool and lightly, but firmly, bandaged from below upwards. Gastric lavage is

beneficial for the associated gastritis, and relieves the distension, which adds to the difficulties of the operation. Subcutaneous salines and glucose restore fluid to the body and carbohydrates to the liver. A blood transfusion may be necessary. Absolute rest is maintained for one hour before operation.

No hypodermic injection is now given before the anæsthetic, although in most of the cases in this series atropine gr. 1/200th was given half an hour before the time of operation. The child wears a woollen vest and a flannel gown, and is carried to the theatre in a warm blanket; one or more well-protected hot-water bottles may be placed between the folds under the child. It is most essential that the theatre and instruments necessary for the operation are ready before the child is brought from the ward, so that there is no waiting. The theatre staff should each know exactly what they have to do, being ready with lotions, porringers, &c., at the right moment to prevent any waste of time. The temperature of the theatre is an important point, and should be between 75° and 80° F. The table should be previously warmed by means of hot-water bottles.

The *anæsthetic* is given in the theatre with the child on the table. The choice of anæsthetic is important.

From 1924 to the end of 1927 six cases were operated on, and in two of them open ether was employed. One developed broncho-pneumonia on the eighth day, but eventually recovered; the other recovered without chest complications. In three cases chloroform and ether were used, and of these two died. One died the day after operation. The vomiting persisted, and at the post-mortem examination a small area of broncho-pneumonic consolidation was present at the right base. The other child died two days after operation; definite signs of consolidation developed at the left base. In one case no note was made as to the anæsthetic employed.

From 1928 onwards seven cases were treated by operation. In all cases gas and oxygen was given through a Boyle's apparatus. In some cases a little ether was added to the mixture. None of the children developed chest complications.

Gas and oxygen is undoubtedly the anæsthetic of choice, and the addition of a little ether at the same time does not appear to increase the danger to any extent. A specially small face-mask is necessary.

THE OPERATION.

Only the site of the operation is exposed. A paramedian incision, 2 in. to 2½ in. long is made high up to the right of the mid-line beneath the costal margin, the advantage of this high incision being that it allows the liver subsequently to protect the abdominal wound; this organ presents itself when the peritoneum is opened, and should be gently lifted up.

The pyloric tumour is delivered, and care is taken to prevent coils of small intestine or of transverse colon from protruding through the wound; the tumour is steadied, and held by the left forefinger and thumb. An incision is made through the peritoneum and superficial muscle-fibres at the junction of the upper and middle thirds of the anterior surface of the pylorus—this being a comparatively avascular area, the meeting place of the superior and inferior pyloric veins. On the one hand this incision extends as far as the duodenum, but not on to it, and, on the other, to a varying extent along the pyloric portion of the stomach. It should be remembered that the pylorus projects somewhat into the duodenum, and great care must be taken not to open the duodenum. The deeper layer of muscle is separated down to the mucous membrane with a blunt dissector or the handle of the scalpel, until the mucosa appears white and glistening and may bulge to some extent. The stomach may now be compressed, and air forced through the pylorus into the duodenum. In this way it may be possible to detect any injury to the mucosa; if the latter has been accidentally incised it must be sutured at once with fine catgut, and if it is thought necessary a piece of omentum sutured over it. The pylorus is returned, and the incision in the abdominal wall should be carefully but quickly closed in layers. The child is carried back to the ward, well wrapped up in a warm blanket, which should also cover the head.

The time taken for the operation was noted in four cases—viz., 6 minutes, 12 minutes, 15 minutes, and 23 minutes; in four, an accidental incision was made in the duodenal mucosa, which was sutured with fine catgut; all four children recovered.

During the operation the cot must be prepared. A hot-water pillow, freshly filled at a temperature of 118° F., is placed under the drawsheet, a folded blanket is in readiness to cover the child, and a small knee-pillow is placed in position. The remaining bedclothes are hung over the electric cradle, protected by a rough brown blanket. The foot of the cot is placed on blocks, and a screen arranged around it. Oxygen and brandy, and the wagon for subcutaneous and rectal salines, must be in readiness.

POST-OPERATIVE CARE.

On return from the theatre the child is placed in the cot, well protected from the light bulbs by the second blanket. The temperature and pulse-rate are taken hourly for 24 hours. If the temperature is rising, the limb bandages are removed. If it continues above 100° F., the electric cradle is removed; if above 101° F., a cold compress is given to the head, and if above 102° F., an ice-bag is applied almost touching the head. The water-pillow is filled every six hours, and feeding is begun four hours after operation. If the condition of the child is poor, rectal saline ℥ i to iii with glucose ℥ss should be given four-hourly, and, if necessary, subcutaneous saline with glucose ℥ iss into each axilla.

Feeds.—Begin four hours after operation.

4th	hr.	after	op.	hourly	feeds	of	1	dr.	for	6	feeds from spoon.
10th	"	"	"	"	"	"	2	"	"	3	" " "
13th	"	"	"	"	"	"	3	"	"	3	" " "
16th	"	"	"	"	"	"	4	"	"	3	" " "
19th	"	"	"	2-hourly	"	"	6	"	"	3	" " "
25th	"	"	"	"	"	"	1 oz.	"	3	Breast.	Test feed.
31st	"	"	"	"	"	"	1½	"	"	6	" " "
43rd	"	"	"	"	"	"	2	"	"	6	" " "

3rd day. Further increase in feeds, return to three-hourly and night intervals, depending on condition of child.

The child is always breast-fed, if possible. Of the ten cases that survived seven were breast-fed.

Temperature.—In all the cases there was a marked rise of temperature immediately after the operation; it was not uncommon for the temperature to rise to 103° to 106° F. in the evening. On the first day a temperature of 102° or 103° F. was usual, rising to 105° F. on the second day. On the third day a gradual fall in temperature began; in two cases it was normal on the second day, and in another two normal on the third day; in the remaining six the temperature gradually subsided, and became normal and steady between the sixth and eleventh days.

Vomiting.—In no case was there projectile vomiting after the operation. In three cases no vomiting occurred at all; in the remaining ten cases there was some vomiting during convalescence. This commonly occurred at two distinct periods: (1) on the first, second, and third day after operation; (2) on the ninth to the fourteenth day. In none of these cases was there any vomiting in the intermediate period or later than the fourteenth day.

Bowels.—Enemata may be required after operation. As a rule they are unnecessary, as the bowels begin to act freely, four to five motions being passed for a few days after operation. On discharge, the child is generally passing two or three motions a day.

Weight and length of stay.—Where convalescence was uninterrupted, in eight cases the average gain in weight on discharge was 15 oz. for an average length of stay of 19 days after the operation. The actual gain in weight varied from 5 oz. to 2 lb., and the length of stay from 15 days to 24 days after operation.

MORTALITY.

Among the 13 cases operated upon there were three deaths.

One child was a very small, undersized, and thin baby of seven weeks, weighing 6 lb. 15 oz.; it had not been breast-fed. On admission gastric lavage was commenced, and continued daily, with some improvement. Operation was performed 32 days later. The anæsthetic employed was chloroform and ether, and the time taken for the operation was 15 minutes.

Subcutaneous salines were given afterwards, but the vomiting persisted, and the child died the next day. Post-mortem, the body-weight was found to be 5½ lb. The pyloric sphincter had been completely divided down to the mucosa, which was found intact and appeared healthy. There was a small amount of clotted blood around the incision in the pylorus. A catheter could be passed readily into the duodenum. There was a small area of broncho-pneumonic consolidation at the right base. All the other organs appeared healthy.

The second fatal case was three weeks old on admission and weighed 7 lb. 12 oz. Operation was performed two days later under chloroform and ether. Death occurred on the second day, signs of consolidation developing at the left base. From the time of the operation until death the child took its feeds regularly and did not vomit.

The third child was three weeks old on admission, and weighed 7 lb. 8 oz. It was noted that the face was a little drawn, the skin somewhat lax, and the fontanelle slightly depressed. Infection of the wound occurred on the second day and suppuration on the fourth day; on the sixth day the stitches gave way, and the wound gaped. Vomiting continued, and wasting set in. The child became collapsed, and died 26 days after operation. No post mortem was granted, but a limited examination of the abdomen was made through the wound. There was a large amount of pus pocketed in the lower part of the abdominal wall, and also in the lower part of the abdominal cavity, shut off by adhesions from the general peritoneal cavity. The lower part of the small gut and greater omentum were involved, and the small gut was distended and inflamed. The stomach was very hypertrophied. The incision in the pylorus was satisfactory, the mucosa being intact and healthy, and there was no sign of inflammation. It is interesting to note that in this case the skin had been prepared with ether and flavine, owing to a rise of temperature following the application of iodine four days previously.

After-results.

Of the five cases treated medically and recovering, three have been traced. In none has there been any vomiting since discharge from hospital. There has been no indigestion or abdominal pain, and the bowels are quite regular. In all three the general health has been excellent. Of the ten cases which survived operation nine are alive and well, but one child died seven months after operation.

He progressed extremely well up to the time of weaning at six months, and weighed 17 lb. He then began to vomit between feeds, diarrhœa set in, and he rapidly went downhill, dying about three weeks later. A note kindly sent from the hospital where he died stated that "on admission he was very ill; eyes were sunken, skin dehydrated, vomiting after feeds but not projectile, stools green; no abdominal tumour felt. The temperature was raised and the pulse was rapid and thready. A diagnosis of infective enteritis was made. The child did not respond to treatment, and died two days after admission. At the post-mortem examination the stomach was rather large, the pylorus thicker than normal; there was a scar ¾ in. long on the anterior surface on its lower half. On opening the stomach some milk was found, but there was no apparent contraction of the pylorus. The stomach mucosa was infected in places, and there were a few adhesions of omentum around the organ; the intestines were apparently normal in appearance."

From the history, it would appear that death was due to infective enteritis, occurring after weaning. The mother sent a photograph of an extremely healthy looking child taken two weeks before death.

The remaining nine cases are strong, healthy children. Of these, seven have had no digestive symptoms at all since the operation. One child suffers from flatulence, and has indigestion on occasions. At the time of weaning he had "bilious attacks" accompanied by vomiting. The other child had attacks of vomiting every two or three weeks for two years after operation; these were accompanied by loss of weight and constipation.

The mothers volunteered the statement that "the children were very advanced for their age," and proudly brought or sent photographs. Most of the children were breast-fed after the operation, and in all the wound had healed well and there were no herniæ. No tumour could be felt in any of the cases.

A comparison of the present weights of 13 children in the series and the weights of average male children of the same age period (Feldman [4]) shows that all except one of the children are over the average weight.

I am indebted to the physicians and surgeons of the Middlesex Hospital for permission to use the notes of cases under their care. My thanks are also due to the ward and theatre sisters for the nursing details.

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CLINICAL AND LABORATORY NOTES

PROLONGED ANURIA IN A WOMAN FOLLOWING PYELOGRAPHY.

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It had been decided to perform pyelography to attempt to discover the cause of abdominal pain, shooting to the left side, accompanied, according to the patient (a woman of 52 years), by some frequency of micturition when the pain was severe. The pain had been troublesome for six weeks. There was no difficulty in the passage of the cystoscope or of the ureteral catheters. The bladder was normal.

Sodium iodide (15 per cent.) was used for the pyelogram; 8 c.cm. were injected into the left pelvis, which was photographed, and then this side was drained, and 6 c.cm. were injected into the right pelvis. The pyelogram showed normal pelves.

Next day the patient passed 25 oz. of urine, slightly blood-stained, and on the second day only 10 oz. were passed. From the third to the seventh day no urine was secreted. During this time she was treated by hot packs, pilocarpine injections, and purgatives; she perspired freely, and her bowels acted well. She felt well in herself until the sixth day when she complained of "heaviness." She vomited small amounts several times each day, but had no headache and was mentally quite alert. On the fifth day the right kidney became palpable. Dry cupping was performed. On the seventh day the left kidney was palpable. The bladder was catheterised on the fifth day but contained no urine. On the seventh day the blood-urea was 122 mg. per 100 c.cm. of blood. Our colleague, Dr. E. G. B. Calvert, who kindly saw the case with us, advised a venesection and accordingly 12 oz. were withdrawn about midday. That evening some six hours later some 6 oz. of urine, deeply blood-stained, were passed, followed by another 6 oz. at 9 P.M. and 4 oz. at 10.30 P.M. On the eighth day she passed 73 oz. containing much blood, and the pathological examination showed pus cells, staphylococci and *Bacilli coli communis* (not a catheter specimen). On the ninth day more than 34 oz. were passed with which was a ureteral clot of

blood some 3 inches long. The blood-urea was now 157 mg.

From now she made an uninterrupted recovery, the urine became clear, and in another ten days the blood-urea had fallen to 29 mg. per 100 c.cm. blood.

Throughout this time the temperature did not rise above 99·2° F. and generally was normal; the pulse-rate varied between 100 and 120 for the first three days, then remained under 100.

We are indebted to Dr. H. H. Sanguinetti for the examination of the urine and estimation of blood-urea.

THE CRITERIA OF CURE IN PULMONARY TUBERCULOSIS.

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The following cases are recorded for their interest in relation to prognosis, and to the definition of "cure" in pulmonary tuberculosis.

Case 1.—A. B. first received treatment in June, 1922, when she was aged 13 years. Her father had recently died of pulmonary tuberculosis, and she came under suspicion on account of a cough and a mild pyrexia. When she was first examined the percussion note was found to be slightly impaired at the left apex, where also the breath sounds were harsh and a few fine râles were heard. Examination of the scanty sputum was negative for tubercle bacilli. She was sent away to a sanatorium, where the pyrexia and râles subsided in a few weeks, and whence she returned home apparently recovered in April, 1923. Her lesion gave no signs of activity during the next five years; she continued to gain in weight, was not troubled with cough, and was afebrile on every occasion but one when she was seen; on that occasion the temperature was 99° F., and she was suffering from coryza. It was thought that she might soon be considered cured. However, in May, 1928, she came up on account of a "cough following a cold," and because she also felt generally unwell. She said that she had coughed up a clot of blood.

Examination of the chest revealed the presence of râles at the left apex, and tubercle bacilli were found in the sputum. A stay in hospital, followed by six months in a sanatorium, brought about an improvement in her condition, but on her discharge tubercle bacilli were still to be found in the sputum. She remained fairly well, and was able to keep on with her work during the next 18 months, but then she began to lose ground again; there were signs of involvement of both lungs, and hoarseness and dysphonia indicated spread of disease to the larynx, in which laryngoscopy revealed unmistakable signs of a tuberculous lesion. She died in July, 1931.

Case 2.—C. D., a magneto repairer, was aged 15 when first seen in March, 1920. On examination he was found to have signs of slight involvement of both upper lobes, some pleural friction was heard low down in the left axilla, and his sputum contained tubercle bacilli. His weight was 7 st. 5¾ lb. He gave a history of having had pleurisy on the right side three years previously. In June, 1920, he went away to a sanatorium, where he stayed until October, 1921. During this time he improved considerably; his weight rose to 9 st. 12 lb.—although, of course, normal growth accounted for some of this—and on return the lesion was apparently quiescent, as the temperature was normal, no râles were to be heard, and there was no sputum. He then returned to work, at which he remained until September, 1930.

When he was seen from time to time at the dispensary there was nothing to suggest activity of his disease, but he occasionally suffered from coryza. He complained from time to time of pain in the right hypochondrium, but this was apparently due to dyspepsia. At the end of 1926 he was told that he might consider that he had recovered, although he should still be careful in his mode of life. His weight was then 10 st. 10 lb. He was a sensible type of patient, and there is no reason to think that he behaved otherwise than carefully.

In March, 1930, he returned on account of pain on the right side of the chest, and cough. There were no adventitious sounds in the lungs, but the temperature was 99° F., and, in April, 100° F. Sputum reappeared, but no tubercle bacilli were found. By the end of April this attack had apparently subsided, he became afebrile, began to put on weight again, and went back to work. By August, 1930, his weight had risen to 10 st. 12¼ lb., which was the heaviest he had ever been. At the beginning of October he suddenly had a hæmoptysis of about half a pint, and took to his bed. During the next few days there were several more hæmoptyses, and signs of broncho-pneumonia appeared, chiefly on the right side, with tubercle bacilli in the sputum. He went rapidly downhill, had several more hæmoptyses, and died on Nov. 17th. At the post-mortem examination old tuberculous lesions with multiple small cavities were found at the apices of both lungs. The pleura was thickened at both apices, and there were adhesions throughout the right pleural cavity. There was a recent tuberculous broncho-pneumonic spread throughout the middle and lowest lobes on the right side, and to a slighter extent in the left lower lobe. The hæmorrhages appeared to have come from the left apex, where blood clot was found.

Case 3.—E. F., was aged 61 when first seen in April, 1925. He was then complaining of a slight cough, which appeared to be due to chronic pharyngitis. There were signs compatible with the presence of an inactive lesion at the apex of the right lung, but otherwise, except for some emphysema, the lungs appeared to be normal. Sputum was scanty, and no tubercle bacilli were found in it. He gave a history that in 1907 he had suffered from tuberculosis of the right lung, and had had part of his epiglottis removed for tuberculous disease. After he had spent six months in a sanatorium his disease was considered to be quiescent. It was not known whether tubercle bacilli had been found in his sputum.

From then until 1918 he remained in good health, and during the war was fit enough to be employed on home service as an officer in the army. In 1918 he had an attack of "pleurisy," from which he apparently made a complete recovery. In earlier life he had been an officer in the Regular Army, had seen service in India and South Africa, and had been a good oarsman and boxer. After leaving the army in 1905 he had gone into business in the City. There was no history of any illness of any significance before 1907. In the winters of 1925–28 he had occasional pyrexial attacks, accompanied by signs of a mild bronchitis and some pharyngitis. No tubercle bacilli were discovered in the sputum, but the organisms most commonly found were *Micrococcus catarrhalis*, pneumococci, and Friedländer's bacillus.

In 1928 he had some signs and symptoms pointing to the possibility of carcinoma of the colon, but a thorough investigation of the alimentary tract failed to reveal any abnormality except colonic stasis. In October, 1929, he was suddenly seized with what was taken to be an attack of influenza. Signs of broncho-pneumonia soon appeared in both lungs; the temperature, pulse-rate, and respirations rose to 102° F., 120, and 36 respectively, and he died on the fifth day of the illness. On this occasion tubercle bacilli were found to be present in the sputum.

Prognosis in tuberculous disease is notoriously difficult; and the would-be prophet has more than once been assured on good authority that he can be certain of only one thing—that his prophecy will be wrong. The diagnosis, and even the definition of "cure," are equally difficult, in spite of all the indications given to the contrary by favourable reports on the various specifics which follow one another in endless succession through brief notoriety to obscurity. The definition officially adopted in this country by the Ministry of Health is that a patient should not be considered as cured of pulmonary tuberculosis until five years have elapsed without any manifestations of active disease. Experience—as witnessed by the first two cases here recorded—shows this definition to be none too stringent. A definition which suggests itself as theoretically admirable—though of doubtful practical value—is that the term "cured" should be applied to the patient who attains the normal expectation of life from the beginning of the quiescent period. Perhaps, in view of the third case recorded above, the only safe prognosis is a variant on that favourite reflection of the Greek dramatists on the insecurity of human happiness—Call no man cured until he is dead.

BRITISH MEDICAL ASSOCIATION.

ANNUAL MEETING IN EASTBOURNE.

SECTION OF MEDICINE.

AT the second session of this section Dr. A. DEANE (Vice-President) took the chair, and a discussion on

Varieties of Secondary Anæmia and their Treatment

was opened by Dr. L. J. WITTS. Analysing the causes of secondary anæmia he emphasised the importance of a group of cases occurring chiefly in women of reproductive age in which none of the usual causes was present, and which formed a distinct clinical entity for which he suggested the name of chronic microcytic anæmia. Twenty-six per cent. of the cases of anæmia admitted to Guy's Hospital during a three-year period he had found to belong to this group, 86 per cent. of these occurring in women. A study of the age-incidence showed a steady increase to a maximum between the ages of 40 and 49, and then a sudden fall. Individual cases might show spontaneous improvement after the menopause. Age- and sex-incidence were, therefore, different from those of pernicious anæmia and achlorhydria. Women affected were found in all classes of the community, but they often showed signs of an asthenic diathesis and gave a history extending back into childhood of intestinal dyspepsia and nervous instability. Clinically, the group was characterised by an anæmia with low colour-index, about 0·5, showing red cells with a diminished diameter and slightly increased fragility. The van den Bergh reaction was negative, the bilirubin and free iron content of the plasma being diminished. The spleen was palpable in one-third of the cases. The bone-marrow showed a normoblastic hyperplasia which on treatment gave way to normal red cell production. Achlorhydria was present in 81 per cent. and glossitis in at least 50 per cent. of cases. These inflammatory changes in the mouth sometimes extended back into the pharynx causing dysphagia. A spoon-shaped deformity of the nails was also common.

The condition, he found, was most likely to be confused with splenic anæmia. The latter, however, was seen commonly in young adults, and showed greater splenic enlargement, while the patient might give a history of jaundice and hæmorrhage. The fingers also showed slight clubbing as opposed to the spoon-shaped nails so characteristic of chronic microcytic anæmia. The frequency of achlorhydria which, as far as his evidence went, was present before the anæmia developed led him to emphasise its possible importance in the ætiology of the disease. He had found massive doses of iron by mouth extremely effective in treating the anæmia. Small doses of iron or injections of iron were useless. He regarded grs. 60 of iron ammonium citrate or grs. 30 of Blaud's pill as minimum doses. Treatment had often to be continued for three months with these large quantities in order to reach a hæmoglobin of at least 80 per cent. and small doses in many cases had to be given indefinitely to prevent relapse. Transfusion was temporarily effective but relapse always followed, and unexplained fatalities might occur in spite of careful direct matching of the blood. Liver, liver extract, desiccated stomach, hydrochloric acid, and splenectomy he had found valueless.

Prof. STANLEY DAVIDSON reported satisfactory results in treating a miscellaneous group of cases with large doses of iron. It had been possible in one case of carcinoma of the colon to bring the hæmoglobin level back to normal, and he treated all cases of secondary anæmia associated with new growth intensively with iron for three weeks before operation, raising the hæmoglobin 30 per cent. in this way.

Dr. JANET VAUGHAN classified secondary anæmias as deficiency diseases dependent upon the lack of those substances essential for normal blood production—i.e., an adequate supply of iron, two factors present in whole liver, one of which was also present in liver and stomach extracts, vitamin C and a factor probably associated with the vitamin B complex. The deficiency might be brought about by faulty diet, failure of absorption or digestion in the alimentary canal, or excessive loss from the body. She quoted her own and other observers' cases to show that idiopathic microcytic anæmia, anæmia secondary to acute and chronic hæmorrhage, and the simple anæmia of infancy responded well to massive iron dosage. She showed that anæmia secondary to chronic hæmorrhage, starvation, deficient dietary in children, past sepsis, and carcinoma was relieved by whole liver combined with adequate iron, anæmia in association with scurvy by vitamin C. The anæmia of chronic myeloid leukæmia had been improved by large doses of arsenic. She emphasised the necessity of adequate dosage of iron and liver, suggesting that Blaud's pill should be given in powdered form to ensure utilisation; grs. 60 of Blaud's pill and ½ lb. of liver a day being minimum rather than maximum quantities. Liver extracts, stomach extracts, and the factor associated with the vitamin B complex had only been found of value in treating macrocytic anæmias.

Prof. H. F. MOORE described a series of cases of idiopathic microcytic anæmia which reacted well to massive iron therapy. He had found achlorhydria common in both diabetes and hyperthyroidism.

In the ensuing discussion Dr. A. V. CLARKE said he had found insulin, 20 units twice a day, in conjunction with glucose, valuable in treating severe anæmia. One case of pernicious anæmia unable to take liver or ventriculin had responded to raw beef incubated with gastric juice. Dr. GEORGE GRAHAM emphasised the value of ferrous as opposed to ferric salts. Dr. E. P. POULTON said that Dr. A. F. Hurst had been the first person to appreciate the value of massive iron therapy in this country. Dr. FREWEN MOOR had found ultra-violet light valueless unless the anæmia improved after the first three applications. Dr. R. C. HUTCHINSON mentioned a case of obstinate varicose ulcer which healed after he had given iron for the associated anæmia, and also a case of high blood pressure associated with anæmia when the blood pressure fell to normal on recovery. Dr. JOHN EASON considered that anæmia associated with syphilis responded well to antisyphilitic treatment. He believed many cases of chronic microcytic anæmia were associated with hypothyroidism. Dr. WILLIAM RUSSELL as early as 1880 had given as much as grs. 180–200 of Blaud's pill with satisfactory results. Dr. R. A. YOUNG instanced cases where small doses of copper gr. ¼ had improved the anæmia. Dr. RUPERT WATERHOUSE asked if there was any knowledge of variations of blood volume in secondary anæmias. Prof. A. RABINOWITZ (New York) said he had found severe anæmia in acute nephritis associated with œdema and in chronic nephritis associated with nitrogen retention. The latter was sometimes macrocytic in character and responded well to liver therapy.

In reply, Dr. WITTS mentioned two cases of simple achlorhydria anæmia which had progressed to pernicious anæmia and one in which the reverse had occurred. He had found the ferrous salts ten times as effective as the ferric salts and that iron in pregnancy anæmia was not always of value. None of the cases in his series had shown signs of myxœdema, the basal metabolic rate being normal. The incidence of microcytic anæmia was, in his opinion, unaffected by environment. He did not believe there were any reliable data concerning blood volume changes in secondary anæmia.

SECTION OF DISEASES OF CHILDREN.

AT the third session of this section, the chair was taken by Dr. E. H. RAINEY, and a discussion on the recognition and treatment of

Birth Injuries in the Newly Born

was opened by Prof. G. B. FLEMING. While emphasising the remarkable recuperative powers of an infant suffering from the traumas of birth as contrasted with its poor resistance to infection, he pointed out that apparently about 30 per cent. of all neonatal deaths were due to birth injury. His paper was based on 350 cases of birth injury among approximately 8000 consecutive infants born alive at the Royal Maternity Hospital, Glasgow. He proposed to devote his remarks only to two groups of cases. Injuries of the infant's *skin and subcutaneous tissues* occurred even in normal labour, and sloughing or infection might cause trouble when the damage to these tissues was more severe. Cellulitis was not uncommon in breech presentations with much bruising and œdema. Injuries to the *nervous system* were the most serious of birth injuries and the most likely to leave permanent disability, though here again the infant's recuperative powers were remarkable. Injuries to the peripheral nerves other than the facial (66 cases in the series) and the brachial plexus (28 cases) were rare; injuries of peripheral nerves of the lower limb might be extremely difficult to differentiate from damage to to spinal cord. Rapid recovery in one to two weeks was the rule in facial paralysis. The early recognition and treatment of brachial paralysis was important, for the earlier the treatment the better the result. Till a satisfactory splint was made the affected limb should be fixed to the pillow with a piece of adhesive plaster round the wrist and a safety pin, the arm being suitably abducted and externally rotated, and the forearm flexed to a right angle and supinated. In very few cases was an attempt at nerve suture advisable. Injury to the spinal cord was commonly produced during breech extractions, and in fatal cases it was usually unrecognised. If the child survived, the symptoms were those of a transverse myelitis. Intracranial hæmorrhage was extremely common; Cruickshank in 800 neonatal autopsies found gross hæmorrhage in 20 per cent. It was extremely difficult to diagnose with certainty during life, but signs pointing to the condition were drowsiness, feeble cry, inability to suck, muscular rigidity, convulsions, bulging fontanelle, and intermittent cyanosis. Dr. Fleming considered that a definitely pigmented or uniformly blood-stained cerebro-spinal fluid with crenated blood-cells was probably the most important manifestation available, but did not afford absolute proof. The fluid should be centrifuged as soon after withdrawal as possible to note any yellow pigmentation. Treatment should be symptomatic: careful nursing, and feeding with the stomach-tube if necessary; if there was increased intracranial pressure lumbar puncture, or in some cases where intraventricular bleeding was suspected, ventricle puncture was required. For convulsions chloral hydrate gr. 1 might be given two-hourly. In 33 cases followed up by Dr. Fleming and Dr. E. D. Morton for a year or more, five were mentally deficient: of these, four had spasticity of one or more limbs, one had spastic diplegia, and one hydrocephalus.

Dr. N. B. CAPON welcomed the choice of birth injuries as a subject for discussion at the section, for it showed that the care of the new-born child was coming into the domain of the pædiatrician. He believed that this would give the infant a better chance, for the obstetrician must of necessity be preoccupied with the mother rather than with the child. The appointment since the war of pædiatricians to the staffs of some maternity hospitals was an important advance. Public attention was at present concentrated on the maternal death-rate, but though the maternal deaths in England and Wales in 1929 were 2787, the deaths of infants in the first month of life numbered over 21,000, and probably over 7000 of these babies suffered from birth injuries of one sort or another. Of the fatal cases of intracranial trauma nearly half had no gross hæmorrhage, and death appeared to be the result of congestion, concussion, or œdema. The clinical differentiation of these different types of trauma had up to now proved very difficult, though the prognosis was not the same. If there was no hæmorrhage and recovery occurred, it would probably be complete; with hæmorrhage there might be after-effects such as hemiplegia or other evidence of permanent damage to the brain, though here again the majority of survivors appeared to be normal. In the differential diagnosis between intracranial trauma and such varied conditions as inanition fever, pneumonia, poisoning through maternal toxæmia, and congenital abnormality of the brain, attention should be directed to the following points: the type of labour, whether the infant is immature, whether the infant shows cyanosis or pallor, failure of the sucking reflex, character of respiration, and abnormal somnolence or excessive crying. The prevention of trauma was a problem for the obstetrician, treatment concerned the pædiatrician. Whether or not lumbar puncture should be done in any individual case was difficult to decide, since on the one hand these infants were often in a state of surgical shock and harm might be done by puncture, and on the other a case of excessive intracranial pressure would die if unrelieved, and the state of the fontanelle did not give the guidance one might expect. He himself used in the past to do more punctures than he now did.

Dr. A. BENNETT agreed about the frequency of intracranial hæmorrhage, but pointed out that the small hæmorrhages discovered post mortem were very often not the cause of death. She suggested the desirability of giving intramuscular citrated blood injections. Dr. DONALD PATERSON and Dr. H. M. M. MACKAY further discussed the difficulties in the diagnosis of intracranial trauma, and also advocated the more extensive trial of intramuscular injections of blood at as early a stage as possible. Dr. C. F. HARRIS inquired whether Prof. Fleming had tried increasing the carbon dioxide intake, combined with extra oxygen, in cases of difficult breathing. If the respiratory centre was injured this method of stimulation of the centre might tide the infant over the first few critical days. Dr. A. A. MONCRIEFF had been experimenting on animals with carbon dioxide and lobeline for the stimulation

of respiration, and considered both agents valuable in the cases under discussion.

Prof. FLEMING replying said he had not seen harm result from lumbar puncture in cases of birth injury. He was against stimulating a damaged respiratory centre by the administration of extra carbon dioxide.

Congenital Pyloric Stenosis.

Mr. C. MAX PAGE gave an account of pyloric stenosis from the surgical standpoint, with details of the operative procedure he employed. He considered the medical pre- and post-operative treatment of primary importance, but found that drugs had little influence on the outcome. He pointed out that though in this country Rammstedt's operation was accepted as the treatment giving the best results, exclusively medical treatment was still widely used abroad without operation, and in one large series treated the mortality was only 14 per cent. Among complications of surgical treatment were: infection of the wound, recurrent vomiting—which should be rare if the operative division at the pylorus was satisfactory—hyperpyrexia, broncho-pneumonia, and gastro-enteritis. In 60 cases operated on by Mr. Page at the Victoria Hospital for Children, the mortality was only 6·6 per cent., whereas with the same operation and, so far as he could determine, the same treatment at St. Thomas's his operative mortality was approximately double.

Dr. NOAH MORRIS gave the biochemical findings obtained by Dr. Graham and himself. In pyloric stenosis, he said, there was an alkalosis with increased blood bicarbonate, and decreased chlorine in the blood and the urine. This alkalosis might occur even if there was no vomiting with the consequent loss of chlorine in the material ejected from the stomach. Correlated with the alkalosis there was depressed and slow respiration. During recovery after operation the carbon dioxide in the blood fell to normal. He suggested that the chlorine in this disease might be largely in organic combination and not combined with a base. His conclusions as regards treatment were that saline solution should be given to supply chlorine, but should not be continued until there was œdema, since this was associated with excessive retention of chlorine; that alkalis should not be given either in the form of citrates or an alkaline stomach lavage, and that if the breathing was slow and depressed a local anæsthetic should be used in preference to a general.

Dr. PATERSON described two unusual cases. In the first, which strongly suggested that hypertrophy of the pyloric muscle occurred during intra-uterine life, a premature baby, born at least one month before term, died ten days later with well-marked hypertrophy of the pylorus. In the second case the infant, died at 8 months old, still with symptoms of pyloric hypertrophy and stenosis, although there had been periods of remission. He pointed out that symptoms usually disappeared spontaneously in babies that survived for 16 weeks. He quoted mortality statistics derived from a series of about 600 cases. The mortality at Great Ormond-street Hospital was roughly about 1 in 4; among 24 cases operated on in private practice there was a mortality of 8 per cent., and in a series of 36 cases treated in one large nursing home there had been not one death. He attributed the great difference in mortality between hospital cases and private cases to the difference in amount of nursing care available for each baby, since the private case had at first a day-nurse and a night-nurse whose whole time was given to its care; and secondly to the fact that in hospital there was always the risk of infection from other children. He outlined his routine post-operative treatment, emphasising the importance of breast milk whenever possible. He advocated operation in all cases of pyloric stenosis when the baby was not gaining weight, unless it was already over 12 weeks old, as at about this age spontaneous recovery might be expected. No cases of hyperpyrexia occurred if atropine was not given.

Dr. DOYNE BELL indicated the danger of too great reliance on radiographic diagnosis in this condition. He had proved that the idea that first-born babies are especially liable to pyloric stenosis was a fallacy. Unlike Dr. Paterson he used very dilute feeds in his post-operative treatment.

Dr. JEAN SMITH corroborated Dr. Morris's findings. The biochemical changes, indicating an alkalosis found in pyloric stenosis were useful in distinguishing this condition from vomiting due to other causes if the clinical findings were not conclusive. If the alkali reserve was not diminished, say in two days, by the administration of saline and glucose, operation should not be further delayed, as such cases do badly on medical treatment.

Mr. R. A. RAMSAY, like Dr. Paterson, advocated local anæsthesia for the operation, and considered a palpable pyloric tumour was of fundamental importance in establishing diagnosis.

SECTION OF NEUROLOGY AND PSYCHOLOGICAL MEDICINE

AT the first session of this section, with Sir JAMES PURVES-STEWART, the President, in the chair, a discussion took place on

Cerebral Hæmorrhage Due to Other Causes than Arterio-sclerosis.

Dr. JAMES COLLIER pointed out that our knowledge and successful treatment of the condition had been greatly increased during recent years, chiefly owing to lumbar puncture and its almost universal application in cases presenting a picture of meningitis, of coma, of apoplexy, or of unexplained headache, and in the routine examination of the cerebro-spinal fluid in disease of the nervous system. The following list comprised the main causes of this type of cerebral hæmorrhage: injury during birth or following concussion, hæmorrhagic pachymeningitis, encephalitis, poliomyelitis, encephalitis lethargica, tuberculous meningitis, sinus thrombosis, abnormal blood states (e.g., leukæmia), anthrax infection, neoplasms, and various aneurysms, such as (*a*) infective, as in ulcerative endocarditis, (*b*) occurring in coarctation of the aorta, (*c*) occurring in polyarteritis acuta nodosa, and (*d*) the berry-like and often multiple aneurysms which have been termed congenital aneurysms. The three most important of these, however, were encephalitis, neoplasms, and cerebral aneurysms.

Many cases of encephalitis which had been confirmed histologically had been complicated by subarchnoid and cerebral hæmorrhage. Encephalitis lethargica was also of interest in connexion with the subject, for many cases of subdural, subarachnoid, and cerebral hæmorrhage from ruptured aneurysms had been recorded in which the clinical picture was hardly distinguishable from that of this type of encephalitis. Neoplasms of soft texture were also important causes of cerebral hæmorrhage, for such a growth might bring on a hæmorrhage even before the appearance of any

local or general signs of tumour. Dr. Collier wished, however, to devote most of the time at his disposal to the subject of cerebral aneurysms. Such aneurysms were held to be of common occurrence, and often gave rise to no trouble during life, since at least one-fifth of the numerous specimens in museums had been chance discoveries at necropsy. They were never syphilitic in origin; the minority were due to bacteriological infections of the blood, such as in infective endocarditis, gonococcal and puerperal infections. In coarctation of the aorta, where the arterial tension above the constricted area in the aorta became extremely high when adult life was reached, the tunica media spread locally under the pressure with the formation of aneurysms, which were often multiple. They were also a feature of the curious malady first described by Kussmaul—polyarteritis acuta nodosa. The cause of the majority of the cerebral aneurysms had not yet been determined with certainty. Eppinger had termed them "congenital aneurysms," implying that they were due to a local congenital defect in the arterial wall. These aneurysms seemed to give rise to no symptoms unless they clotted up, calcified, or became adherent to surrounding structures—usually to some of the cranial nerves—or unless they ruptured. They might rupture in any situation within or upon the brain; they then produced the syndrome of a rapidly severe apoplexy, with blood in the cerebro-spinal fluid. These aneurysms had a tendency to rupture slightly and to leak slowly, after which they often healed only to leak again later on—sometimes in another situation. They might leak into the subdural space, and there form an organising hæmatoma, with a clinical picture sometimes resembling encephalitis lethargica. They often leaked into the subarachnoid space, and then most commonly produced a syndrome precisely resembling meningitis; the distinction from the latter condition was the presence of blood in the cerebro-spinal fluid and the relief produced by draining. The transient phenomena which might follow on a not too rapid and not too copious hæmorrhage upon or within the brain were very remarkable, and some of them were likely to give rise to difficulty in diagnosis, as they might occur without headache or signs of meningeal irritation. The more common of these transient signs were attacks of unconsciousness, lasting from a few hours or many days, headache resembling migraine, hemiplegia, aphasia, convulsions, delirium, pyrexia with headache, unconsciousness with albuminuria or glycosuria. The following was a short list of the regions in which an aneurysm might burst or leak: (1) transdural rupture—aneurysm of the posterior communicating artery; (2) subdural rupture; (3) subarachnoid rupture; (4) intracerebral rupture, either from a surface aneurysm, which became adherent to the brain tissue or from an aneurysm buried in the brain tissue; (5) primary rupture into the ventricle; (6) rupture of an internal carotid aneurysm into the cavernous sinus—the clinical picture being that of a unilateral pulsating aneurysm.

The treatment of subarachnoid hæmorrhage was an interesting matter for discussion, since it had been held that the repeated draining away of the effused blood in lumbar puncture increased the tendency to bleeding, though it undoubtedly relieved the symptoms for the time being. Dr. Collier's practice was always to drain freely and repeatedly so long as the symptoms of increased intracranial pressure persisted, and he had never had cause to regret this practice.

DISCUSSION.

Prof. A. J. HALL limited his remarks to subarachnoid hæmorrhage. There were several interesting features in these cases to which he would call particular attention. (1) Occasional massive albuminuria. In one of his cases in which this condition was present the kidneys were found to be normal on post-mortem examination. (2) The occurrence of Korsakow's syndrome during recovery. Although the symptoms might persist for several weeks, recovery was the rule. (3) The frequency of slight or severe exacerbations after the initial hæmorrhage. This might occur owing to the true nature of the illness not being recognised at first and complete rest not being insisted upon; on the other hand, it might also occur with the patient at complete rest in bed. (4) The occasional occurrence of convulsions during recovery. Sometimes a sudden muscular effort might determine the moment of leakage, but in others the precipitating factors were so slight as to justify the term "spontaneous hæmorrhage." Dr. Hall then cited, in illustration, certain cases of intracranial aneurysm resulting in hæmorrhage. There was also an important medico-legal aspect, in that if the leak was initiated by some strenuous act during the course of employment, or a blow, as when boxing, what should be one's attitude in assessing compensation, or in attributing the result to the blow?

Dr. W. J. ADIE stated that the symptoms of subarachnoid hæmorrhage might appear without evidence of previous ill-health in patients with polycystic kidney and infective endocarditis. He thought that aneurysms leaked most often in warmer weather, and in several of his cases a diagnosis of "sunstroke" had previously been made. Premonitory headaches, resembling migraine, were common, and might occur at intervals for many years; in their most characteristic form the hemicrania was always on the same side of the head. Retinitis proliferans sometimes yielded evidence that an aneurysm had leaked in the past, and gave the clue to the cause of the symptoms in a later attack. He had also seen bilateral hæmorrhage into the vitreous, and also three cases in which herpes zoster was a complication. Recovery from the mental symptoms resembling Korsakow's syndrome was always complete.

Mr. HUGH CAIRNS discussed the surgical treatment of intracranial hæmorrhage, and described cases both of subdural and intracerebral hæmorrhage which had been treated successfully by operation. He emphasised the importance of small bilateral subdural explorations under local anæsthesia in every case in which subdural hæmatoma was suspected. A subdural hæmorrhage might not necessarily be localised, and might resemble a subarachnoid hæmorrhage, blood trickling down subdurally into the spinal canal, and even yielding pure blood on lumbar puncture. Mr. Cairns also described a case in illustration of the good result that could be obtained from the removal of large blood clots from within the brain itself.

Dr. J. PURDON MARTIN confined his remarks to hæmorrhage in relation to the dura mater. Widespread subdural hæmorrhage might be a sequel of head injury without any fracture of the cranium; it might prove fatal, or give rise to chronic subdural hæmatoma. The localising signs of the latter were often vague and deceptive. Often the blow causing the injury appeared trivial, especially in elderly subjects, and might consist merely in jerking the head. There was often a latent period of weeks or even

months, or only vague and indefinite symptoms before signs of cerebral compression made their appearance. Sometimes the extravasated blood did not clot, but appeared to suffer defibrination.

Dr. J. G. GREENFIELD considered only that form of cerebral aneurysm which had been called "berry aneurysms." In his experience these aneurysms had been found most frequently in the arteries of young or middle-aged subjects who had shown either no trace of atheroma or only small isolated plaques. An abnormal arrangement of the cerebral or systemic arterial system had been commonly described in patients dying from ruptured intracranial aneurysm, a fact which constituted one of the grounds for considering a congenital malformation of vessels as the cause of these aneurysms. In an important paper published in November, 1930, Wiley Forbus had given a clear description of the true defect. He had shown that it was primarily a defect in the muscular coat of the artery rather than in the elastic coat, and had demonstrated its presence not only at the point where the aneurysm formed, but at practically every bifurcation of the cerebral vessels in these subjects. He had demonstrated such a defect also in the mesenteric vessels. Dr. Greenfield had examined the cerebral arteries in five non-atheromatous cases of cerebral aneurysm, and had found this defect present in all, whereas it was not present in two cases of intracerebral hæmorrhage of the atheromatous type. As regards treatment, the value of drainage of cerebro-spinal fluid in cases of subarachnoid hæmorrhage had been stressed by Dr. Collier, but there were some who were still opposed to this procedure on theoretical grounds, holding that reduction of intracranial pressure removed some support from the outer surface of the aneurysms. This theory had been proved to be fallacious by new knowledge on the relationship between the intracranial arterial pressure and the general intracranial pressure. Baillard and Magidot had demonstrated the fact that the pressure in the intracranial arteries rose and fell with raising and lowering of the general intracranial pressure. It had been known since the work of Cushing that rises in the general intracranial pressure up to the normal diastolic blood pressure did not cause any rise in the latter as measured in the brachial artery. But such rises automatically caused corresponding rises in the cerebral arteries, so that, whereas the pressure in the retinal artery was normally 50 per cent. of that in the brachial artery it rose to 60 or 90 per cent. of it with increase of intracranial pressure. Berens had also shown that, in even so small a branch as the retinal artery, the systolic pressure is from eight to ten times the general intracranial pressure as measured by the lumbar puncture manometer. It was therefore evident that: (1) the rise of intracranial pressure which was caused by rupture of a basal aneurysm automatically raised the blood pressure within that aneurysm, and was therefore likely to cause further bleeding; (2) that reduction of the general intracranial pressure by drainage of cerebro-spinal fluid would automatically reduce the pressure inside the aneurysm to a far greater extent than it would remove support from outside it.

Mr. A. A. MCCONNELL described a case of subdural hæmatoma in a man aged 70, in whom headache appeared two days after jerking the head. In the course of a week focal symptoms—paresis of the left arm and leg—made their appearance. At no time was consciousness impaired, although cerebration was slow. Operation revealed a definite subdural hæmatoma, and the patient made a good recovery.

(*Continued at foot of next column.*)

PLASMOQUINE IN MALARIA.

A REVIEW OF RECENT EXPERIENCES.

PLASMOQUINE, the trade name for N-diethylamino-isopentyl-8-amino-6-methoxy-quinoline, was introduced into the therapeutics of human malaria in 1926, and for five years workers in various parts of the world have been testing its uses and limitations.

The Army in India.

There is no organised body of men whose efficiency has been more handicapped by malaria than the British Army serving in India. Accurate statistics of the morbidity from this cause have been kept over a long period, so that it has become possible to estimate the value of any form of antimalarial treatment with greater degree of accuracy amongst soldiers under continuous and disciplined observation than amongst a similar body of civilians. An exhaustive trial in India of plasmoquine and quinine has recently been reported [1] by Major J. A. Manifold in the treatment of benign tertian malaria, the form of malaria most prevalent amongst serving units and most detrimental to service. In this paper Major Manifold has drawn on the accumulated experience of 325 publications on this subject which have appeared lately. From a study of these it has been realised that many of the untoward symptoms hitherto described were due to the slender margin between the therapeutic and toxic doses of plasmoquine. Sinton, Smith, and Pottinger having in 1930 reported unfavourably on a daily dose of 0·06 g., it was resolved to try 0·04 g. plasmoquine plus 1·25 g. (grs. 20) of quinine taken for 21 consecutive days. The troops concerned in mass treatment included both British and Indians, the latter varying greatly in physique. The experiment being spread over such a huge area of country with varying degrees of humidity and temperature, the conditions varied enormously. In most stations it was quite impossible during the malaria season to discriminate between fresh

[1] Jour. R.A.M.C., May–June, 1931.

(*Continued from previous column.*)

Mr. ASTLEY CLARKE mentioned hilarity as an early, and almost only, symptom in a male case, aged 24, of aneurysm of the basilar artery, which ruptured and caused death. He had also seen two other cases in females, in whom laughing of an unusual character was observed as an early symptom in similar intracranial hæmorrhage.

Dr. HINDS HOWELL emphasised the frequency with which subdural hæmorrhages were bilateral, and cited an instance in a man aged 60. Following what at first appeared to be a mild head injury, left hemiplegia developed after six weeks. Operation was performed on the right side, and a large subdural hæmatoma was found and evacuated. The patient improved for 24 hours, after which he relapsed into a semi-comatose condition. The operation area was re-explored, as it was thought that further hæmorrhage might have occurred. Nothing was found, and it was hoped that the compressed brain still remained unexpanded. The patient died on the following day, and autopsy showed another subdural hæmatoma on the opposite side. The speaker had seen cases of subarachnoid hæmorrhage with massive albuminuria similar to those described by Prof. Hall, and there was an obvious danger of making a preliminary diagnosis of uræmia. In association with the albuminuria, glycosuria and acetonuria might also occur.

cases of malaria and relapses. Any attempt to take into account different factors by varying the dosage of plasmoquine was obviously impracticable; no difference was made in the dosage used for individual cases and any fresh attack within six months of completion of treatment was considered as a relapse. By entrusting the treatment however to a large number of medical officers in different parts of India many individual results and opinions could be obtained. Sufficient plasmoquine was available to treat 4000 cases, and in each command certain officers were selected to undertake the treatment and to keep records of the cases treated. In stations in which a laboratory was situated the medical officer carrying out the treatment worked in close coöperation with the officer in charge of the laboratory.

The treatment of all cases of benign tertian malaria consisted of one tablet containing 0·02 g. plasmoquine combined with grs. 10 of quinine in the morning and a similar amount in the evening, the total course of treatment being 21 days. In all, 3187 cases (1286 British, 1901 Indian) of benign tertian malaria completed this course and are included in the analysis. Cases treated after Dec. 31st, 1929, and subtertian cases treated partially with quinine and subsequently with plasmoquine were not included. A special watch was maintained for toxic symptoms and precise instructions having been issued regarding their nature. In the great majority of instances these symptoms were never severe and frequently were not mentioned in the hospital register after the first 20–30 cases had been treated. The effects of suggestion, particularly as regards "colic," could not be neglected for it was noticed that once patient and medical officers had gained confidence in the drug, complaints became fewer and eventually ceased altogether. As a corollary to this the larger the number of cases treated in a hospital, the more favourable the opinions expressed on the value of the treatment and the fewer were the toxic symptoms of real importance noted—a statement which tallies with the experience of other workers both here and in the tropics.

TOXIC SYMPTOMS.

The percentage of toxic symptoms recorded, such as abdominal pain, cyanosis, and digestive disturbance was less amongst Indian than amongst British patients, owing no doubt to the greater attention paid to mild symptoms. In view of the fact that 99 per cent. of all the cases ultimately completed their course of treatment, toxic symptoms cannot have been severe. Abdominal pain constituted the one constant complaint; it was found to occur with less frequency when the drug was taken on a full stomach or with a large draught of water. The incidence of this pain definitely increased with the duration of the treatment indicating, as had already been realised, that plasmoquine is a cumulative drug showing its maximum effects on about the sixth day of treatment. Epigastric pain occurred as a rule within a few days of beginning the course and disappeared quickly on temporary cessation of plasmoquine administration. In this series of cases cyanosis is recorded as having been noted in 4·3 per cent. of British and 0·67 per cent. of Indians. The lower proportion amongst the latter group is probably accounted for by the difficulty of recognising it in dark-skinned races. As previously noted by Fischer and Weise the cyanosis becomes evident clinically about the seventh day and it appears also to occur with greater frequency in anæmic cases and is accompanied usually by epigastric pain. Many cases appeared to lose their pink complexion during treatment, but yet could not be called actually cyanosed. It seems then that in a small percentage of cases there does exist a definite individual idiosyncrasy to the action of the drug with reference to epigastric pain and cyanosis. In the average case of cyanosis a continuance of the treatment does no harm and the methæmoglobinæmia does not increase.

Opinions were much divided as to the effect of the treatment in the reduction of chronic enlargement of the spleen; only in acute enlargements is it effective in reducing the organ to normal size. As regards the pyrexia it was found that on the whole patients responded more rapidly to treatment with plasmoquine and quinine than to quinine alone, and in no case was it necessary to give intravenous or intramuscular quinine. Relapse is the crux of efficient treatment and out of this trial the test has emerged triumphant. The relapse-rate was surprisingly small, the average for all classes and stations being 5·2 per cent. If cases relapsing after what may be roughly called the end of the malaria season in the various stations only were accepted as relapses, then the relapse-rate would be as low as 2·4 per cent. The figures for the relapse-rate are definitely favourable as compared with the usual quinine treatment. In the quinine-treated control series of Sinton, Smith, and Pottinger at Kasauli the relapse-rate was as high as 42 per cent.

There were two deaths amongst the Indian cases. The first occurred after vomiting and was not considered to be due to plasmoquine treatment; the second appears to have been due to blackwater fever (methæmoglobinæmia) and it is possible that it was a case of mixed injection with benign and subtertian malaria. There were two other cases of mild methæmoglobinæmia which recovered; they may have been mild cases of blackwater fever more likely than instances of toxic plasmoquine methæmoglobinæmia.

The report closes with quotations from various medical officers in the I.M.S. and R.A.M.C. On the whole the opinions were favourable and encouraging. The efficacy of this treatment combined with simplicity of administration and shortness of course appealed to both medical officers and troops. Most questions on this important subject having been satisfactorily answered it remains to be ascertained whether plasmoquine can be safely issued to patients not under medical supervision. The answer would appear to be definitely in the negative, but if the margin of safety can be made greater and good results obtained by utilising 0·03 g. plasmoquine as a daily dose plus quinine (grs. 20) for 21 days, a definite advance would be made. Investigations on these lines are being carried out at the malaria treatment centre, Kasauli. There 57 cases have been treated up to date. Toxic symptoms have been non-existent and the patients have been able to play an occasional game of hockey or football.

Central America.

Three other reports of importance are before us. The staff of the United Fruit Company have been assiduous in recent years in investigating plasmoquine treatment. In the nineteenth annual report of the company's medical department, Dr. N. P. Macphail writes on plasmoquine as an aid in malaria prevention in Guatemala. The conclusions arrived at as the result of the administration of 1,388,000 tablets of plasmoquine were as follows: Plasmoquine effectively destroys the gametocytes of all forms of malaria when given in doses of 0·03–0·04 g. daily for one week. Combined

with quinine, plasmoquine is a safe and sure way of preventing the formation of gametocytes during an attack of malaria; and the combination destroys all types of parasites. Toxic symptoms need not be feared when the dosage is limited to 0·03 g. daily for a week or more. It is considered that there is ample clinical proof to justify the hypothesis that plasmoquine is of importance as a curative factor in chronic cases which have resisted extended efforts at eradication with quinine and arsenical preparations.

Dr. Vincente Bustillo, at Banes, Cuba, has come to much the same conclusions. The doses of plasmoquine in his practice are much smaller and consist of one plasmoquine tablet (0·01 g.) for four days and grs. 20 of quinine. Children under 5 years of age received 0·005 g. for eight days and no untoward effects with the dosage above mentioned have been noted. Since 31 out of 34 cases showing crescents in the blood became negative within nine days subsequent to the initiation of plasmoquine treatment, it can be concluded that the doses used were sufficient to cause a disappearance of gametocytes from the peripheral circulation from one to eight days in 91 per cent. of the cases and within nine to twelve days in the remaining 9 per cent. The easy disappearance of gametocytes leads to the belief that plasmoquine has a direct action upon them; but it is probable that they are devitalised to be afterwards destroyed by phagocytoses.

In assessing the rôle of plasmoquine in the sanitation of Panama, O. T. Brosius exercises due care in the field of its application. Working with a force of employees totalling some 4800 the difficulty of gauging the value of "blanket treatment" with plasmoquine and quinine may be imagined. The conclusions arrived at are that plasmoquine has probably played a more active rôle in effecting a reduction in malaria infections than was given credit for in earlier attempts to determine its exact value in tropical sanitation. It is emphasised that with a distinct reduction in malaria, there was a simultaneous decrease in the hospital admissions, which was more or less in direct ratio to the malaria decline. In a community such as Panama with a comparatively limited turnover in the labour force by sufficiently frequent plasmoquine and quinine blanket treatments the sterilisation of the gametocytes to malaria carriers can be repeated at given intervals.

The Present Position.

It is evident from the information summarised above and the publication[2] of the prophylactic experiments of S. P. James and his co-workers that the whole question of plasmoquine has entered a new phase. The progress so far achieved has been satisfactory, but a less poisonous compound than plasmoquine would undoubtedly lead to greater confidence in its extended usage away from medical supervision, and it is much to be hoped that in the future less toxic compounds will be found. More information is needed about the effect of administering plasmoquine under all sorts of conditions. Recent work has confirmed in a striking manner the accuracy of the pioneer observations made with a limited series of cases at the Tropical School in Hamburg, and subsequently at the Hospital for Tropical Diseases, London.

[2] THE LANCET, August 15th, p. 341.

REVIEWS AND NOTICES OF BOOKS

Heart Disease.

By PAUL DUDLEY WHITE, M.D., Instructor in Medicine, Harvard Medical School; Physician, Massachusetts General Hospital, Boston. London: Macmillan and Co., Ltd. 1931. Pp. 931. 42*s*.

Dr. Paul White's book is the most comprehensive treatise on cardiology which has appeared in the English language in recent years. It touches on every aspect of the subject, from the most important to the most trivial, and therein lies its chief defect—namely, a lack of perspective. There is too much detail, too little emphasis on some of the main points. To deal adequately with all the subjects to which the author refers would require a book of at least twice this size, and it is doubtful whether any one cardiologist would be competent to write such a book. Possibly this criticism will be less pertinent for the American than the English reader, for there is a wide difference in the outlook on medicine in the two countries. In America the investigation of clinical cases follows a more universal plan than in Britain; every fact is carefully recorded in the case-notes, with the result that sometimes it appears to us difficult to sort out the capital matter. With us the investigation of cases is less systematic. Our idea of a great clinician is a man who can pick out the essential points of a case and give his undivided attention to them alone, discarding everything which is irrelevant. The great teachers of medicine in this country have impressed on the minds of their students vivid pictures of individual cases in the wards. This difference of viewpoint is strikingly illustrated by Dr. White's book. He refers occasionally, it is true, to the occurrence of particular symptoms in patients in his own practice, but from beginning to end his treatment of the subject is strictly systematic.

The book is divided into four parts, which deal respectively with the methods of investigation including symptoms and physical signs, the ætiological types of heart disease, structural lesions, and lastly, disorders of function. Part I. includes an excellent account of cardiovascular radiology, an aspect of diagnosis which has hitherto been scantily treated in most English text-books. In other parts of this section the author is apt to over-classify. The statement that such and such a condition may be due to 1, 2, 3, or 4 is a joy to students, but is too watertight for the more advanced reader. In the chapter dealing with sphygmomanometry (p. 127) the author refers to the disappearance of the Korotkow sounds as the diastolic end-point. With this neither physiologists nor clinicians will agree.

Part II. opens with some extremely interesting graphs comparing deaths from heart disease in the United States with those from cancer and tuberculosis, and the age-incidence in fatal cases. But influenza and focal infections, which we regard as important causes of cardiac invalidism, are allotted only half a page each, while gout is dismissed in two lines. Sense of proportion is similarly lacking in the reference to Raynaud's disease and chilblain (Part IV.), and to angina pectoris, to the consideration of which the author devotes only 20 pages. Part III. deals with structural lesions. This inevitably leads to

difficulties. Why for example refer at all to "the course, prognosis, and treatment of cardiac enlargement" (p. 468) which obviously varies with the cause of the lesion? And why consider under the heading of aortic valve disease two such entirely dissimilar conditions as syphilitic aortitis and rheumatic endocarditis, or why group together syphilis and atheroma of the aorta? Maybe Dr. White has adhered to an older classification, feeling that in recent years the pendulum has swung too far and morbid anatomy is being neglected at the expense of disorders of function. With this attitude most cardiologists will heartily agree, but the remedy does not lie in merely reverting to the faults of the older classification.

In his general outlook in cardiology Dr. White favours the middle course, as may be seen from his frequent use of the phrase "the truth probably lies between these two extremes," but from time to time he is lured back to the camp of the anatomical diehards. This may be the reason why in enumerating the causes of congestive heart failure (p. 565) he gives first place to valvular defects and includes auricular fibrillation as one of the less common causes of the condition. In England it would be no easy matter to collect a hundred cases of congestive heart failure with normal rhythm. The section dealing with the treatment of congestive heart failure, and especially digitalis therapy, is excellent and full of common sense though exception might be taken to the statement (p. 567) that cardiac dyspnœa is due primarily to oxygen lack.

The book is well got up. The illustrations, many of which are taken from the works of Sir Thomas Lewis, are clear and well produced. An attractive feature is the detailed quotations from the writings of pioneers in cardiology. In his preface the author states that the bibliographic index is intended to be as brief as is consistent with its usefulness. Nevertheless it occupies nearly 200 pages, and gives some indication of the amount of work the author must have put into his book. British cardiologists will find in Dr. White's book a good systematic account of heart disease based on views accepted in great American schools.

Recent Advances in Radiology.

By PETER KERLEY, M.B., B.Ch. (N.U.I.), D.M.R.E. (Camb.), Assistant Radiologist, Westminster Hospital; Radiologist, the Royal Hospital for Diseases of the Chest. London: J. and A. Churchill. 1931. Pp. 324. 12*s.* 6*d.*

THIS volume of the Recent Advances series is written in a clear and convincing style and Dr. Kerley deserves congratulation for having steered his way so skilfully through many of those stretches of radiology that are still uncharted. Sometimes, however, he errs on the side of a dogmatism that is not justified. His insistence on the basic pathology is commendable in a book which is, as it should be, clinical in character.

The general outline and construction of the work are satisfactory, and it is well indexed. On the whole it may be taken as a sound guide, though in certain details the teaching would not be generally accepted. Many hypotheses are set down as facts, as, for example, the statement that "the arch of the aorta is always dilated if aneurism is present in another part of it," or that "we now know Perthes' disease, Kienböck's disease, Köhler's disease and Schlatter's disease to be manifestations of the same disease." The author's gift for clear exposition has led him to make pictures appear more definite and sharply defined than the facts justify, especially in connexion with differential diagnosis. The novice reading this book would see little difficulty in making a definite diagnosis in cases where experience would advise great caution, with efforts to obtain confirmation from every possible source before an opinion is expressed.

Discounting this tendency, which is manifest throughout the book, the volume provides a good résumé of present-day knowledge, especially as regards the German point of view. The names of German workers are freely given to various signs and methods. For instance, the absence of evidence of a gastric peristaltic wave as it passes over an affected area of diseased wall is here called Fraenkel's sign, the reference given being 1927. This phenomenon was noted many years ago in some of the earliest papers on the subject, and there seems little need to associate it with any observer's name. The short chapter on radiotherapy sacrifices accuracy to brevity, and would have been better omitted. The book is well and clearly written and is likely to achieve success. It will be of great value to radiologists, particularly those of experience who can discount the author's enthusiasm and appreciate the vast amount of work that has been embodied in this book.

The illustrative radiograms and line drawings are very clear. Many of the former are touched up to emphasise the points, and although the immediate purpose is served, the novice may carry away a false impression of the appearances he is likely to discover in his own films in similar conditions.

Piersol's Human Anatomy.

Ninth edition. Under supervision of G. CARL HUBER, M.D., Sc.D., Professor of Anatomy and Dean of Graduate School, University of Michigan. London: J. B. Lippincott Company. Pp. 2104. 45*s.*

WE welcome this new edition of a well-known book, which has long passed beyond the region of general criticism. In this issue the new matter has to do mainly with histological questions, such as the origin of blood-cells and like matters, and the B.N.A. has been introduced throughout. Care has been taken to preserve the pagination of the last edition, so that guides and references can remain unchanged. Several new illustrations have been introduced, and the work in general has been brought up to date.

Rest and Pain.

By JOHN HILTON, F.R.S. Edited by W. H. A. JACOBSON. London: G. Bell and Sons. 1931. Pp. 514. 5*s.*

Hilton's famous lectures on the influence of mechanical and physiological rest in the treatment of surgical diseases were published some 70 years ago, after their delivery before the Royal College of Surgeons of England. In these the insistence on the diagnostic value of pain is associated with elaborate knowledge of surgical anatomy, and the popularity which the work obtained is not surprising. During Hilton's life the book was several times revised, but after his death his editor left the text untouched, and between 1880 and to-day 14 editions have been published. Though modern knowledge has made much of the book out of date, its general messages and its philosophy remain just. It is one of the works that promise to earn and keep the over-used epithet of "classic."

THE LANCET.

LONDON: SATURDAY, SEPTEMBER 5, 1931.

THE COMMON COLD.

THE medical profession is much abused because it cannot cope with the common cold. Here, people say, is a world-wide disease which is always with us and is responsible for an enormous amount of discomfort and loss of human efficiency; yet nothing is known about its causation, and its treatment is empirical and unsatisfactory. To anyone acquainted with the facts such a criticism is of course manifestly unjust. The problem of ætiology is far from being a simple one; the very fact that it has remained unsolved so long is proof enough of that. None of the ordinary laboratory animals is suited for its investigation and the disease is so widespread in man as to make him of little value as an experimental animal unless he is subjected to most rigorous quarantine and irksome isolation.

For some time now the idea has been current among bacteriologists that the primary cause of the common cold is not one of the ordinary bacteria, but a member of the group of filter-passing viruses. Support to this view was lent by the findings of W. KRUSE (1914), G. B. FOSTER (1916), and H. DOLD (1917); but doubts have now been settled beyond all dispute by the masterly investigation which Prof. A. R. DOCHEZ and his colleagues describe elsewhere in this issue. Their work soon led them to the conclusion that the visible bacteria present in the nasopharyngeal discharge were only of secondary importance, so they turned their attention to the filter-passing viruses. And since observation had shown that the chimpanzee was subject to a respiratory infection resembling in every way the human cold, they used this animal for their experimental work. The results of these experiments on apes left no doubt that the cold virus was a filter-passer. Typical colds were produced in chimpanzees by means of the filtered nasopharyngeal washings from human cases, passages in series from ape to ape were realised, and infection by contact was shown to take place. Needless to say the most stringent precautions were taken to exclude infection from outside sources. DOCHEZ and his colleagues then proceeded to carry out the same experiments with human volunteers. Here again the greatest care was taken to ensure that their subjects were not infected before being taken on experiment and that all outside sources of infection were excluded. The human experiments were just as conclusive as those made with the chimpanzee. But this is not all. Using a culture medium consisting of a special buffered broth containing chick embryo tissue and brought to a suitable reduction potential by means of cystein hydrochloride, these workers have been able to cultivate the virus of the common cold. And with the fifteenth subculture, representing a dilution of 1 in 1,000,000,000,000,000, they have produced typical colds in two out of three human volunteers.

Not less interesting are the speculations of DOCHEZ and his collaborators on the part played by this virus in respiratory infections in general. Their experimental work has shown them that this virus renders the respiratory tract more susceptible to the implantation of pathogenic organisms, as well as enhancing the activity of any potential pathogens which may be present there. Observations in America have shown that the common cold has three peaks of incidence, one in September and October, another in January and February, and a third in April and May. Further, the cases occurring in winter and spring are usually more severe than those arising in the autumn. Coincident with this increased virulence of colds as the winter lengthens into spring there is an increasingly wide distribution of such organisms as the pneumococcus, the influenza bacillus, and *Streptococcus hæmolyticus* in the nasopharynx of healthy individuals. Thus as the winter advances the opportunity enlarges for these potential pathogenic organisms to cause infection, with the result that their distribution becomes more widespread and their virulence is probably enhanced. It is at this period of the year that pneumonias and bronchial infections are most common, and it may well be that DOCHEZ'S virus is in part responsible for this. Be this as it may, the principal thing for the moment is that the ætiology of the common cold has at last been cleared up. Complete confirmation of these findings is forthcoming from an investigation carried out by P. H. LONG and a team of collaborators[1] on human volunteers at the Johns Hopkins University and an important field is opened up for further work. All who have followed these researches of DOCHEZ and his associates must have been struck not only by their thoroughness, but also by their undeviating march towards success. It is a brilliant piece of work.

THE BASEMENT CHILD.

FROM the Bishop of SOUTHWARK we have in a small volume[2] some penetrating observations upon the social conditions of one district of his industrial diocese. He records the circumstances of living which are compulsory upon the inhabitants of the southern shore of the Thames from Battersea to Woolwich, an area that is densely inhabited by a wage-earning class—where, that is, employment is obtaining a return in wages. Dr. GARBETT gives a vivid picture, based upon knowledge and checked by experience, of an urban area, where men, women, and children are condemned to make such homes as they can amid the evils of overcrowding—an area where the safeguards of sanitary supervision and the benefits of social service, both well displayed, cannot keep the menaces

[1] Jour. Exper. Med., 1931, liii., 447.

[2] In the Heart of South London. By the Right Rev. Cyril Forster Garbett, Bishop of Southwark. London: Longmans, Green and Co. 1931. Pp 152. 3s. 6d.

of ill-nutrition at bay. And the reason is the abominable housing, where basements are always damp and living rooms often so overshadowed by high walls that direct light cannot penetrate them.

The text of the book was expounded by his lordship recently in the Upper House, when in an eloquent speech[3] he drew attention to the large number of persons now living in underground buildings, not only in South London, or London generally, but in all our big cities. He inquired if the local authorities were playing their part in the construction of new houses to meet the evils of overcrowding, and brought forward a motion for the institution of a central housing council, which, he considered, would stimulate the erection of suitable habitations for the working classes. This motion he withdrew later, on an explanation from Lord SNELL, speaking for the Government, that the powers possessed by the local authorities were already adequate, and the provisions of the Slum Clearance Act of last year were quoted in support of his words. And although Lord SNELL deprecated concentration upon a particular aspect of the evils which had to be dealt with, the Bishop of SOUTHWARK'S indictment of basement dwellings made a valuable impression upon the House. And now Dr. GARBETT'S words receive powerful endorsement in the report for 1930 of the school medical officer of the London County Council, which appeared this week, for, in a section of his general annual report,[4] Dr. KAY MENZIES includes the results of a special inquiry made by Dr. A. BANKS RAFFLE into the condition of children living in basements, the information being compiled by the inspectors of the Council, and derived from elaborate inquiry among families occupying basements in eight metropolitan boroughs, widely separated in geography, and differing in rateable values.

The boroughs selected were Kensington, Paddington, Westminster, Islington, Finsbury, Poplar, Stepney, and Southwark. The children were examined in their schools, and the findings were checked by double control, each basement child being compared with two non-basement children of the same sex and age, and, as far as possible, in similar social circumstances. In this way 254 basement children were examined and 508 controls, and the examiner was throughout in ignorance which of the little subjects were basement dwellers. The inspection cards revealed that the incidence of pneumonia and bronchitis was not markedly different in the two classes, but there was a definitely higher incidence of diphtheria among the basement children and a greater tendency to rheumatism. The physical examinations were directed especially to debility, defective sight, rheumatism, and rickets. At most ages, and particularly among the older children, the basement children compared unfavourably with the controls in weight and measurement; they were as clean and no more anæmic, but they were not as well-nourished. While defective vision was present in similar proportion—about 24 per cent.—symptoms of rheumatism were nearly three times as frequent among the basement children, and throat and nose affections were found in 7·4 per cent. of the basement children and in only 3·3 per cent. of the controls. Rather unexpectedly rickets was evenly distributed, at just above 7 per cent., between the two classes. At first sight the contrasts revealed do not seem to justify the universal indictment of the basement as a home for children, but Dr. RAFFLE points out wisely that the figures do not tell the whole story. He supports the Bishop of SOUTHWARK'S words by saying that in some areas, giving Southwark as the example, it was possible to pick out, on sight, some of the basement children from their controls, while inquiry into educational standards showed that children living in basements do not, as a class, progress at school satisfactorily—the head teachers returning as definitely backward 24·8 per cent. of the controls and 43·6 of the basement children, who, moreover, were double as irregular in attendance. This inquiry may not reveal the lurid conditions expected in some quarters, but it shows enough to give cause for thought to the public authorities concerned. It is the ambition of the financier to get in on the ground floor, but it should not be that of the tenement dweller, whatever the pecuniary inducement.

[3] THE LANCET, July 18th, 1931, p. 163.
[4] Annual Report of the London County Council, 1930, vol. iii., Part II.

PINK DISEASE.

THE disorder of childhood to which this name among many others has been ascribed has definite and striking clinical characteristics,[1] but an ætiology and pathology which have not yet been clearly established. For many years it has both fascinated and perplexed pædiatricians, and lately the interest has not been limited to medical circles. The development of our knowledge of the disease, its discovery by independent observers in different countries, and the various titles it has received, furnish material for an interesting study. It is difficult to say whether or not it is a new disease, but the fully developed clinical picture is so characteristic that it seems impossible that it should have passed unrecognised for long, and in this country it is generally conceded that it is a "symptom complex" which was not met with before the late war. Certainly there is no published reference to it before February, 1914, when H. Swift, of Adelaide, reported to the tenth Australasian Medical Congress at Auckland, 14 cases of what he thought was a new disease, and called "erythrœdema." Although this paper constitutes the first printed account, the condition was noticed in Australia many years before, for A. J. Wood,[2] in 1921, reporting a series of 91 cases, remarked that he had seen examples of the disease at Melbourne for 30 years previously, and that even before that time W. Snowball of the same city had recognised it and spoken of it as "raw beef hands and feet." C. B. Clubbe had also observed the disease in Sydney for several years before this

[1] THE LANCET, 1921, i., 871.
[2] Med. Jour. Austral., 1921, i., 145, see THE LANCET, loc. cit.

date, and is responsible for calling it "the pink disease," a name which possesses advantages over many of its synonyms in that it accurately describes the chief symptom, is not misleading, and does not commit the user to any particular view of its pathology. The condition also seems to have been recognised in Germany and France some years before it was recorded, for in a paper published by P. Selter[3] in 1926–27 he states that in 1903 he reported eight cases under the title of "trophodermatoneurose" or "vegetative neurosis" to a medical society at Cassel, in Germany; and P. Haushalter,[4] of France, writing in 1925 on a "neurovegetative syndrome in infants," remarked that he had been familiar with the syndrome since 1911.

In the United States of America the disease seems first to have made its appearance about 1915, and in Canada in 1918, although its identity with the pink disease of Australian writers was not recognised until some years later. In 1917 A. H. Byfield, in a paper read by title before the American Medical Association, described the earliest cases as "trophoneuroses probably due to an infection," and again in 1920 as "a polyneuritic syndrome resembling pellagra-acrodynia (?) seen in very young children."[5] A little earlier in the latter year J. B. Bilderback had recorded some instances of an unknown disease which he had met with in his practice; later W. Weston[6] came to the conclusion that these were examples of a disease which assumed epidemic proportions in Paris in 1828–29, and to which the name of acrodynia was given by Chardin. In 1923 it was quite clearly pointed out by J. Zahorsky that all these American cases were identical with pink disease. Although Weston and many other American authorities still regard pink disease as a juvenile form of acrodynia, it is highly improbable that the two diseases are identical, one reason being that acrodynia was an epidemic disorder attacking adults, whereas pink disease is a sporadic disease of infancy and early childhood; further, Petren has produced good evidence to show that acrodynia was due to the drinking of wine contaminated with arsenic. In England the first case of pink disease was described in 1921 by Parkes Weber, who demonstrated it before the Dermatological Section of the Royal Society of Medicine as "a condition somewhat resembling lupus pernio in a child"; in 1922 H. Thursfield and D. H. Paterson[7] published a report of a case under the heading of "dermatopolyneuritis"; and in 1923 L. G. Parsons[8] reported six cases, two of them observed in 1920. E. Feer,[9] of Zürich, was the first continental writer on this disease, and in 1923 he redescribed the condition as a "neurosis of the vegetative nervous system," pointing out that many of the characteristic symptoms were referable to a disturbance of the sympathetic nervous system—e.g., extreme irritability, sweating, cold and clammy extremities, tachycardia, raised blood pressure, salivation, hypotonia, photophobia. This communication created such an impression that on the continent the disorder is usually referred to as "Feer's disease."

An annotation in our issue of May 2nd summarised some of the latest necropsy findings, and W. G. Wyllie and R. O. Stern[10] have just published an important paper recording the pathological findings of seven further cases. Degenerative changes were found in the peripheral nerves of four of them; chromatolysis of the central type, more apparent in the lumbo-sacral cord than elsewhere, was present in the anterior horn cells in five; and in all cases the spinal cord showed that diffuse infiltration with small cells which was first described by J. G. Greenfield and Paterson. These results, they state, confirm those already recorded and afford further evidence that lesions in the nervous system are to be found in a large proportion of cases. Taking all the published morbid findings into consideration it must be admitted that as yet no single lesion has been established as a constant characteristic of the disease, and it is a point of some significance that the clinical symptoms have always been considerably in excess of the pathological findings. It is therefore not surprising that the ætiology of pink disease has given rise to much discussion. Three theories of its causation have been propounded: First that it is a deficiency disorder; secondly that it is the result of an infection; and thirdly that it is a dysfunction of the vegetative nervous system. The resemblance to pellagra, which was commented upon by the earlier American writers, is a point in favour of the deficiency theory, and some transatlantic authorities still believe that the two diseases are related. There are, however, essential differences between them, perhaps the most obvious being the absence in pink disease of the seasonal variations of the rash which are so characteristic a feature of pellagra. Again, a deficiency in any of the known vitamins has never been demonstrated in the diets of sufferers from pink disease. On the other hand, Marshall Findlay and R. O. Stern[11] found that young rats developed a clinical condition which resembled pink disease, and pathological changes in the peripheral nerves, and a cellular infiltration of the cord similar to that described in autopsies on pink diseases, when they were fed on a diet which contained all the known vitamins but in which the sole source of protein was egg white. Moreover, this condition was cured by the addition of dried yeast or raw liver to the diet, which suggested that it was due to a deficiency in some dietetic factor but not one of the known vitamins. Applying these findings to children suffering from pink disease, Wyllie and Stern report the recovery of five patients fed on a liver diet, and they consider that there is a dietetic factor contained in liver which has a definite curative value.

[3] Arch. f. Kinderh., 1926–7, lxxx., 244.
[4] Rev. Neurol., 1925, i., 401.
[5] Amer. Jour. Dis. Child., 1920, xx., 347.
[6] Arch. Pediat., 1920, lvii., 513.
[7] Brit. Jour. Child. Dis., 1922, xix., 27.
[8] THE LANCET, 1923, i., 490.
[9] Ergeb. d. inn. Med. u. Kinderh., 1923, xxiv., 160.
[10] Arch. Dis. Child., 1931, vi., 137.
[11] Ibid., 1929, vi., 1.

The not infrequent occurrence of prodromal catarrhal symptoms in connexion with the nose and throat; the increased incidence shortly after the last great pandemic of influenza; and the appearance of acrodynia in 1828, exactly one year after the first influenza pandemic of the nineteenth century, have all been stressed as points in favour of the infective theory. M. Péhu and A. Mestrallet believe that the condition is the result of a specific infection of the nervous system, closely analogous to encephalitis, and both H. Janet and R. J. Turquety maintain that it is due to a neurotrophic virus which gains access to the nervous system through the naso-pharynx. The pathological evidence in favour of the infective theory is, however, but slight. Finally Feer's suggestion that the condition is a functional disorder of the vegetative nervous system rests on the clinical evidence of such a disturbance, and the fact that in one fatal case he could not find any alteration in the sympathetic system after a very careful examination. However true it may be that such a disorder of function is the cause of the symptoms, this theory does not provide any explanation of the functional disturbance itself. What evidence there is as to the cause of pink disease appears to favour a dietetic deficiency as *an* ætiological factor, although possibly not *the* factor. Indeed it may well be that, as first suggested by Byfield, the disease, or the dysfunction of the vegetative nervous system which produces it, is due to a combination of the first two causes—in other words, that a dietetic deficiency may produce a state which favours an infection.

ANNOTATIONS

FOOD-POISONING IN CALIFORNIA.

AT one time California had an unenviable notoriety for outbreaks of botulism, but vigorous action has reduced these to small proportions. The three examples of food-poisoning now reported from this State by Prof. A. J. Geiger and co-workers [1] were due to salmonella organisms. In the first, at Sacramento in January, 35 persons were visited with diarrhœa, vomiting, and the usual acute symptoms. The incubation period was very short, being two and a half to four hours, but there were no deaths. The sufferers belonged to a party of 60 who attended a late evening banquet. While a considerable number of foods were eaten, inquiry showed fairly definitely that the main meat course of chicken and chopped-up veal, with cream sauce, was at fault. From this *Bacillus enteritidis* was isolated, but with great difficulty, partly, it seems, because the bacilli were very scanty and partly because the mixture was heavily contaminated. The premises were rat-infested, and the authors consider that rats were the probable source of the infection. The mixture was given ample time to be infected, for the chickens were cooked and boned on Jan. 18th, left about overnight, and mixed with the veal on the 19th, and the mixture with cream sauce was warmed up on the 20th to be served at the late supper. Another outbreak was at a hospital, and 200 people were afflicted with somewhat similar symptoms to those of the Sacramento outbreak. The incubation period was also short, two to four hours, and again there were no deaths. While the food involved seems definitely to have been rice pudding covered with a fruit sauce—since this was the only food eaten in common—the evidence does not clearly show the course of infection. A *B. aertrycke* strain was isolated from the rice pudding, and was not found in the original rice. Only one stool of a sufferer was examined, and that revealed nothing, while no agglutination blood tests were carried out. There is no actual proof therefore that the patients suffered from a living infection with this organism. The pudding was heated in a steam cooker for about an hour, and then kept for six to eight hours before being served. Possibly infection took place subsequently, but apart from an unconfirmed suspicion that a commercial rat-killer had been employed, no source of infection was found. No information is given about the source of the milk used.

The third outbreak, which was in March, was also in a hospital, and caused the illness of 52 persons. Here, too, there was a short incubation period (three to four hours), the symptoms being nausea, vomiting, diarrhœa, and great prostration. The peccant food was an egg soufflé shrimp mixture, served with tinned peas; it was harmless when first eaten, but after remaining overnight in the kitchen it became definitely poisonous on being warmed. *B. paratyphosus* A was isolated from a stool from the assistant cook, and the outbreak is ascribed to this organism; but there is insufficient warrant for this. Three excreta samples were negative, and in the single fatal case the internal organs disclosed no food-poisoning bacilli such as are to be expected almost invariably when death is due to a living salmonella strain. No blood agglutination tests are recorded, and none of the food mixture eaten was available for bacteriological examination.

Apart, however, from obscurities about the exact bacterial strain responsible, and whether the illnesses were due to living bacilli or to their toxins only, the three outbreaks illustrate the fact that food-poisoning is usually due to manipulation of food, which gives opportunities of infection, and that the greatest danger comes from meat or milk foods which are heated and then left about to get cool. The greatest attention must be paid to the storage of foods which have been heated and are allowed to cool slowly, and this especially applies to those which are good culture media for the food-poisoning bacilli. Such attention, together with scrupulous cleanliness, will prevent a considerable proportion of outbreaks, and perhaps the lesson is being slowly learnt. In Great Britain food-poisoning has been relatively commoner than in the United States, but of late it has seemed to be diminishing.

[1] Public Health Reports, Washington, July 3rd, 1931, No. 27, xlvi., 1565–72.

BLOOD TRANSFUSION IN STREPTOCOCCAL SEPTICÆMIA.

THE treatment of streptococcal septicæmia by immuno-transfusion, as advocated by Almroth Wright in 1918, is the subject of a study by Prof. P. Abrami and Dr. A. Tzanck in *Paris Médical* for August 1st. After discarding some 29 cases, because sufficient data were not available, they confine their observations to

43 cases of streptococcal septicæmia in which a hæmolytic streptococcus was either actually found in the blood (22 cases) or the case was recognised as such on circumstantial evidence. Those in which it could not be found in the blood were not as a class any more benign than the blood-positive cases, among which there were 12 recoveries. Among the blood-negative cases there were nine recoveries. The donors were sought as far as possible among women who had recovered from puerperal fever, and had been inoculated according to the technique described by Jaubert in 1928. Further, whenever it was possible, the donor was reinoculated with autogenous vaccines, made from the patients themselves within six hours of the transfusion. The cases recorded in some detail emphasise the point, made by the authors early in their paper, that the measures adopted in each case were as varied as were the processes which led to recovery in such a goodly proportion of their cases. It follows that their study is not of the kind that lends itself to summary review.

INTRACRANIAL TUMOURS.

THE last 20 years have seen considerable advances in our knowledge of intracranial tumours from their clinical, pathological, and therapeutic aspects. The time is therefore ripe for a critical review of this subject and Dr. F. M. R. Walshe's paper [1] is a valuable survey of this wide and ever-widening field of medicine. It was impossible within the limits imposed to deal with the whole of our present knowledge of intracranial tumours, and Dr. Walshe has necessarily confined himself to what may be termed the growing points of the subject. The clinical and therapeutic importance of the pathology of tumours of the brain was hardly recognised at all until comparatively recently. Chiefly owing to the researches of Cushing and his pupils it has been learned that the different types of tumours, even the different types of glioma, often exhibit a characteristic age-incidence and rate of growth and a predilection for certain situations in the brain. Hence it is becoming possible for the clinician with increasing frequency to diagnose not only the presence and situation of an intracranial tumour, but also its precise pathological nature, which proves of importance in forming an accurate estimate of its prospect of removal, of the peculiar difficulties likely to be encountered in the task, and of its probable malignancy. The importance of pathology is reflected in the fact that Dr. Walshe devotes more than a third of his paper to the classification of intracranial tumours. He gives an admirable account of the development of the modern conception of the gliomas as tumours corresponding in structure to different stages of development of embryonic neural tissue, and though he does not commit himself to the finality of any existing classification, he fully recognises the importance of current theories.

Dr. Walshe analyses the pathological physiology of raised intracranial pressure and rightly lays stress upon the importance of generalised epileptiform fits as an initial symptom of tumour. Except among neurologists it is inadequately recognised that "generalised epileptiform fits making their first appearance in an otherwise healthy subject in the fourth or fifth decades of life should at once raise a suspicion of intracranial tumour." In considering some localising symptom-complexes he devotes special attention to tumours in the neighbourhood of the optic chiasma, whose early recognition is of great importance for the preservation of vision. He is refreshingly critical of current loose conceptions of pathological sleep and "the sleep centre."

Any impartial worker cannot fail to be impressed with the revolution in the diagnosis of intracranial tumours which has been brought about by radiography. Not many years ago it was held to be useless to radiograph the skull in cases of suspected tumour because no abnormality was likely to be found. Since then we have learned that even if the tumour itself is invisible valuable clues as to its presence and situation are often provided by alterations in the bones of the skull, and the intraventricular or intrathecal injection of air has in many cases established the localisation of a growth when clinical methods have failed to do so. Dr. Walshe puts the case for the use of these accessory methods of investigation very fairly, rightly emphasising that they are supplementary to clinical methods. Advances in the surgery of the nervous system do not come within the scope of his review and little is as yet known concerning the value of therapeutic irradiation in the treatment of tumours of the nervous system. The important principle which underlies any method of treatment of intracranial tumour is that it must take full account of the pathological nature of the growth.

[1] Quart. Jour. Med., 1931, xxiv., 587.

THE NEW CONVENTION ON NARCOTIC DRUGS.

THE conference on the limitation of the manufacture of narcotic drugs, which sat at Geneva from May 27th to July 13th, under the presidency of Mr. de Brouckère, of Belgium, concluded a new convention which is intended to "supplement" the Opium Conventions of 1912 and 1925 by seeking to render more effective the international control of the traffic in drugs of addiction. It will be remembered that the Geneva Convention of 1925, while reciting that "results of great value" had been produced by the original Hague Convention of 1912, nevertheless deplored that "the contraband trade in, and abuse of, the substances to which the convention applied still continue on a great scale," and accordingly sought "to complete and strengthen the provisions" of the earlier treaty. The draft of the new convention has now been published, and it may be well to consider in what respects it indicates an advance upon the international obligations already entered into by the high contracting parties.

The new convention, unlike those of 1912 and 1925, has little concern with raw opium or prepared opium, and deals mainly with morphine and cocaine and their derivatives. Article 2 defines "the drugs" to which the provisions are applicable, and the handiwork of the experts who advised the conference is evident in adding to morphine, diacetylmorphine, and cocaine and their salts the following: dihydrohydroxycodeinone, dihydrocodeinone, dihydromorphinone, acetyldimethylodihydrothebaine, dihydromorphine, their esters and salts. In a subgroup appear ecgonine, thebaine and their salts, benzylmorphine, and the other esters of morphine and their salts; while in a separate group are placed methylmorphine (codeine), ethylmorphine, and their salts.

Each contracting party is to furnish annually to the permanent central board estimates of the drugs required, distinguishing the amounts for use for medical and scientific purposes (including export), for conversion, for reserve stocks, and for Government requirements. The seven chapters and 34 articles of the new convention amount, in fact, to an elaboration

of the provision contained in Article 9 of The Hague Convention, which reappeared as Article 5 of the Geneva Convention of 1925, which was as follows:—

"The contracting Powers shall enact pharmacy laws or regulations to limit exclusively to medical and legitimate purposes the manufacture, sale, and use of morphine, cocaine, and their respective salts, unless laws or regulations on the subject are already in existence. They shall coöperate with one another to prevent the use of these drugs for any other purposes."

The foregoing restriction was also to apply to medicinal opium, to heroin, to all preparations containing more than 0·2 per cent. of morphine or more than 0·1 per cent. of cocaine, and to all new derivatives thereof and to other alkaloids of opium which may be shown to be liable to similar abuse.

The estimates furnished by the contracting parties are to be examined by an advisory body of four persons appointed respectively by the League's advisory opium committee, the central board, the health committee of the League, and the Office International d'Hygiène Publique. The estimates, with any explanations that may be vouchsafed, and any observations of the supervisory body, will be supplied to all the Powers concerned. Manufacture of any of the drugs in excess of the estimates is not to be permitted, due regard being had to imports and exports. Export of heroin, except to countries which do not manufacture it, and then only on import certificate and consignment to government, is forbidden. New drugs derived from opium or coca are not to be manufactured unless the government concerned is satisfied of their medical value; and if the health committee finds they are drugs of addiction they shall be subject to the provisions of this convention. Preparations containing less than 0·2 per cent. of morphine and less than 0·1 per cent. of cocaine, and preparations of codeine and ethylmorphine (with some reservations) are to be brought under control. Every year the permanent central board is to report for each country the estimates for each drug, together with the amounts consumed, manufactured, converted, imported, and exported. Explanations vouchsafed by any contracting party of failure to carry out his obligations will also be published, but not "in such a manner as to facilitate the operations of speculators or injure the legitimate commerce of any high contracting party." Each party is to create a "special administration" for applying the provisions of the convention, supervising the trade in drugs and "organising the campaign against drug addiction." Strict supervision over "raw materials" is to be required and drugs seized in illicit traffic are to be destroyed, converted into non-narcotic substances, or appropriated to medical or scientific use under government control. Particulars of each case of illicit traffic are to be communicated, through the secretary of the League of Nations, to the other parties to the convention.

The chairman of the conference claims that it has "succeeded," but it has to be remembered that it will not come into force until "90 days after the secretary-general of the League of Nations has received the ratifications or accessions of 25 members of the League of Nations or non-member States, including any four of the following: France, Germany, United Kingdom of Great Britain and Northern Ireland, Japan, Netherlands, Switzerland, Turkey, and United States of America." In the final act attached to the convention the governments are invited to consider the desirability of setting up State monopolies over the manufacture and trade in narcotic drugs, but the German delegation dissented from this proposal. A suggestion is also made as to the possibility of replacing heroin by a drug of less dangerous character. Finally an approximate estimate of the annual world requirements is tabulated as follows: morphine, 9 tons; heroin, 2 tons; cocaine, 5½ tons.

GANGRENOUS APPENDICITIS.

It is gradually being recognised that there are two types of "acute appendicitis," one which without early operation rapidly goes on to perforation and general peritonitis, and the other which often subsides without surgical intervention, although it may be followed by recurrent attacks. This fact has led to the practice by some surgeons of the "delayed treatment" of acute appendicitis. D. P. D. Wilkie [1] has adduced evidence to show that the former type is primarily not an inflammation but an acute obstruction, and that all cases of gangrenous appendicitis are the sequel of an obstruction of the lumen, usually by a fæcolith. He states that while acute obstruction of the appendix is, without surgical treatment, a most deadly disease, acute inflammation of that organ is comparatively harmless. LaRoque [2] now puts forward a claim for the value of a sign, first noted by R. Brittain in 1928, in the diagnosis of gangrenous appendicitis. This sign is elicited by pressure on the right lower quadrant of the abdomen, especially at McBurney's point, when the right testicle is immediately drawn upwards. As a rule the testicle remains retracted until the pressure is withdrawn, it then returns to its original position. Occasionally, however, the testicle may drop back although pressure is maintained, only to retract again momentarily as the pressure is removed. Since the sign has been present in nearly 500 cases of gangrenous appendicitis, and has not been found in a series of more than 300 cases of other acute abdominal conditions, LaRoque regards it as pathognomonic of gangrenous appendicitis and an indication for immediate operation.

CARE OF THE INSANE IN IRELAND.

The irony of Dean Swift's end has often been commented on, but it is seldom recognised that when "he gave the little wealth he had to build a house for fools or mad" he was endowing the first public hospital in Ireland to be wholly devoted to the care of the insane. In an interesting paper,[3] written for annual meeting of the Royal Medico-Psychological Society in Dublin, Dr. Kirkpatrick has described the scanty, and often barbarous, provision that existed prior to 1735, when Swift resolved to settle "his whole fortune to erect and endow an hospital for the support of idiots and lunatics." The matter went forward slowly. At first the mayor and aldermen of Dublin were named as trustees, but presently "upon the City's favouring Fanatics" the will was altered, and it was not until May, 1740, that it was signed in its final form. On August 16th, 1742, a writ *de lunatico inquirendo* was issued, and returned the next day, finding the Dean of St. Patrick's a person of unsound mind; three years later he died. In 1757 his intention was realised and the hospital declared open. For the next 50 years St. Patrick's continued to be the

[1] Bristol Med.-Chir. Jour., 1930, xlvii., 103.
[2] LaRoque, G. P.: Amer. Jour. Med. Sci., August, 1931, p. 191.
[3] A Note on the story of the Care of the Insane in Ireland up to the End of he Nineteenth Century. By T. Percy C. Kirkpatrick, M.D., Litt. D. Dublin: The University Press. 1931.

only hospital of its kind in Ireland, though the Houses of Industry in Cork, Dublin, Limerick, and Waterford sheltered many lunatics, along with prostitutes, incorrigible offenders, vagabonds, and others. In 1810 the Bloomfield Retreat was opened by the Quakers, following the example of their kind in York, and in the same year the Government made the necessary grant for building the Richmond Asylum; in both of these institutions humane and enlightened methods of treatment were employed from the first. During the next half century much was done, largely by Act of Parliament, so that in 1851 there were 11 district asylums, besides a number of private mental hospitals, so well managed as to earn the praise of Conolly of Hanwell. By 1900 the number of district asylums had been doubled and there was steady advance in many directions.

In Ireland, as elsewhere, the history of the care of the insane is for the most part a painful story of insensibility and cruelty, now happily ended; but in this case it is illuminated at its beginning by the remarkable regulations of the ancient Brehon laws, codified about the time of St. Patrick, in which appears an enlightened concern about the duties of the healthy community towards those "on whom the maddening wisp had been thrown," to use the old Irish phrase. The sagacious rules there laid down to prevent the insane from having offspring are nowadays of particular interest.

MENINGITIS DUE TO THE SPIROCHÆTE OF JAUNDICE.

LATELY several cases of meningitis in Paris have been traced to infection with the spirochæte of Weil's disease (*Leptospira icterohæmorrhagiæ*). In some of them there was no jaundice, and the diagnosis was suggested by the occupation of the patient, and proved by the high titre of the antibody reaction given by the patient's blood. One which is typical of the condition is described by Harvier and Wilm.[1] It was that of an exhibition diver at a restaurant, who became ill in December with evident signs of meningitis. Lumbar puncture gave an opalescent fluid with 350 cells per c.mm., 95 per cent. of which were polymorphonuclear leucocytes; it contained no visible bacteria, and cultures, on ordinary media, remained sterile. The patient recovered after repeated lumbar punctures in ten days' time, but five days later a second rise of temperature occurred, this time without meningeal symptoms. Schwarz, who has collected seven other non-jaundiced cases, describes the condition as an acute form of meningitis with severe contractures of the legs, but without herpes, petechial rash, or conjunctival congestion. The cerebro-spinal fluid may be opalescent on the sixth to the eighth day of the disease, if the meningeal reaction is severe, in which case the cells are chiefly polymorphonuclear, or it may be clear with a cell count either of mixed type or principally mononuclear. In other cases the meningitis has been associated with inflammation of other serous membranes—pleural, peritoneal, and synovial—which has cleared up without leaving any trace. The condition is one which might easily pass undiagnosed unless the possibility of spirochætosis were kept in mind, and the fact that infection by the spirochæte may produce symptoms other than jaundice is of importance from the point of view of public health.

[1] Bull. et mém. Soc. méd. des hôp. de Paris, 1931, xlvii., 893.

PYREXIAL TREATMENT OF CHOREA.

SOME two years ago a drug called Nirvanol was introduced for the treatment of chorea.[1] Eight to 14 days after administration is begun it produces a reaction in the form of fever, rash, general malaise, and an eosinophilia. Treatment is immediately suspended, and the chorea improves as the reaction subsides. Occasionally, however, the sequels of the reaction are not so happy, as is emphasised by F. Goebel,[2] who has seen purpura hæmorrhagica, nephritis, and a fatal case of dermatitis exfoliativa following the exhibition of nirvanol for chorea. He points out that although this drug is effective in treating the disease, it cannot be recommended with confidence, since it may actually cause death, and such a catastrophe can at present be neither foreseen nor prevented. Most observers think that the improvement in the chorea is not directly due to the fever so produced, since similar improvement may follow apyrexial reactions. L. P. Sutton,[3] however, now describes 25 cases of chorea in which rapid and striking amelioration followed pyrexial therapy. In the first case the fever was the result of accidental overdosage with phenobarbital, and the improvement which followed prompted the further investigations. The other 24 cases were treated by intravenous injections of typhoid-paratyphoid vaccine administered daily for as long as six or seven days, the dose being graduated so as to produce a febrile reaction lasting six to seven hours with each injection. Under this régime the average duration of symptoms after the onset of treatment was eight to nine days. The advantages claimed for this method are that it causes fever immediately, is capable of producing it in any patient, and is not complicated by rashes or other disturbances.

ENCEPHALITIS FOLLOWING VACCINATION OR EXANTHEMS: A REQUEST TO PRACTITIONERS.

THE Ministry of Health has issued a statement asking medical practitioners to help in maintaining full investigation of nervous manifestations (meningitis, encephalitis, polio-encephalitis, &c.) developing within four weeks of vaccination. A similar request in 1926 provided material for the consideration of the Rolleston committee. Having issued two reports [4] the committee has now ceased work, but particular study of these rare cases is still required. Furthermore, the Ministry now wants information about cases of acute disease of the nervous system following acute infections, such as measles, German measles, chickenpox, whooping-cough, or influenza; for the Rolleston committee noted that such conditions bear a close pathological resemblance to post-vaccinal encephalitis and demand continued study and comparison. Communications of the kind requested, if received in time, will permit of local inquiry to ascertain clinical details which may not subsequently be obtainable, and will sometimes enable the department to arrange for expert post-mortem examination and pathological examination of specimens. Practitioners are therefore asked to report without delay every case of disease of the central nervous system with an acute onset within four weeks of vaccination or immediately following an acute infectious illness. The intimation

[1] See Poynton, F. J., and Schlesinger, B. E.: THE LANCET, 1929, ii., 267.
[2] Deut. Med. Woch., July 31st, p. 1313.
[3] Jour. Amer. Med. Assoc., August 1st, p. 299.
[4] Report of the Committee on Vaccination. Cmd. 3148. See THE LANCET, 1928, ii., 233. Further Report of the Committee on Vaccination. Cmd. 3738. See THE LANCET, 1931, i., 85, 97.

should be sent to the Senior Medical Officer, Med. I., Ministry of Health, Whitehall, London, S.W.1.

The statement adds that the Lister Institute has prepared an antivaccinal horse serum, doses of which can be supplied on application to the Ministry. It also calls attention to a reference, in the report of the Committee on Vaccination, to the success which has been obtained in certain cases by the treatment of post-vaccinal encephalitis by intravenous injection of serum derived from persons who have been recently vaccinated.

EARLY CARDIAC FAILURE.

WHEN there is a long history of diminished cardiac reserve, and one finds localised or generalised œdema, evidence of stasis in the liver and lungs, and definite pathological signs in the heart itself, the diagnosis of cardiac failure is simple. But this is an end-result, an acknowledgment of defeat by the patient and by his doctor, and to-day, when the prevention and early diagnosis of disease is rightly emphasised, and when heart failure is still responsible for the deaths of many men and women at the height of their mental abilities, we must focus attention chiefly on the early condition. A recent review by Prof. L. Bogendörfer [1] shows that the greatest importance, as Mackenzie taught, is still to be attached to an accurate history; it is essential at the same time to make a close study of the patient's mentality, for otherwise symptoms due in reality to early cardiac failure may easily be attributed to neurosis. Bogendörfer describes tests for the estimation of cardiac function, but since Nature knows no average, attempts to estimate cardiac reserve by counting the *absolute* increase of the pulse-rate after exercise such as knee-bending are likely to fall to the ground. The same applies to blood pressure and the quality of the pulse; the latter is often used to gauge the power of the heart, but actually it depends not only on the volume per beat of the heart, but also on many arterial factors. Repeated examination of the same patient, however, may be very valuable. Less often emphasised, but well worth attention are minor disturbances of water metabolism. The heart normally works with part only of its available power; with increased needs reserve powers which the healthy heart can easily and rapidly supply are called into play. With ordinary daily wear and tear the reserve power of the heart becomes insufficient, and water tends to be stored in the body by day, only to be excreted at night. Hence the importance, as Quincke pointed out, of nocturnal enuresis as an early symptom. The body, Bogendörfer points out, can retain considerable amounts of fluid without the development of visible œdema, and even 4–7 litres accumulated may give rise to no signs of œdema or transudate into the body cavities. This latent œdema, he says, can only be unmasked by the regular use of the weighing scales and with the estimation of intake and output of fluid. Kauffmann has shown in apparently fully compensated heart disease that raising the end of the bed will result in increased secretion of urine; 5 oz. of tea are given hourly to the patient lying flat in bed and the bladder is emptied hourly; after four hours the foot of the bed is raised and the patient is kept in this position for two hours, urine again being passed hourly. Small amounts of rather more highly coloured, acid urine, with a higher specific gravity, perhaps containing a trace of albumin, are passed in the first period; an increased amount of urine of low specific gravity passed in the second period is a certain sign of latent œdema. Attempts have also been made to estimate cardiac reserve by determining how long a patient at rest can hold his breath after full expiration; he then is exercised by a quick walk and again holds his breath after full expiration. The time taken depends partly on volition, but also to a large extent on the rapidity with which carbon dioxide accumulates in the blood.

[1] Zeit. für Ärztl. Fortbild., May 1st, p. 288.

SCARLET FEVER IN INDIA.

IT is not so many years ago that enteric fever was supposed to be rare amongst Indians. This belief, owing to blood tests and more frequent post-mortem examinations, is now exploded, and it appears that the impression, widespread until recently, that scarlet fever is almost if not entirely non-existent amongst Indians, must go the same way. It is at any rate seriously challenged [1] by Dr. P. T. Patel, who is in charge of the principal infectious diseases hospitals in Bombay. Few physicians can have practised for many years in India without occasionally coming across a sporadic case having all the classical features of scarlet fever, but such cases are almost always in Europeans, and it is usually impossible to trace their origin. Leonard Rogers remarked (1919) that scarlet fever occurs in India as an imported infection, that there is no evidence that it is an indigenous disease, and that it rarely if ever attacks Indians. This is the view that has hitherto been generally accepted. From time to time, however, cases resembling scarlet fever have been reported in Indians, and in 1920 K. Macleod came to the conclusion that the disease did affect Indians, though showing no disposition to become epidemic among them. Since then isolated cases have been repeatedly observed in Bombay and elsewhere, and Dr. Patel is now definitely of opinion, from his own observation, that scarlet fever is indigenous in India, and he quotes many cases in support of this view. The suggestion is made that climatic conditions have modified the disease, and it is clear that the rash may easily be overlooked on a dark skin.

[1] Indian Med. Gaz., June, 1931.

ARISTIDES AGRAMONTE.

Dr. Agramonte, who has just died of heart disease at New Orleans at the age of 62 years, was the last survivor of the four actors in a brilliant episode in the story of yellow fever. With Walter Reed, James Carroll, and Jesse Lazear, he was sent by the Surgeon-General of the United States Army to Cuba in June, 1900, to investigate the epidemic of yellow fever then raging there. The sequel is well known. Two of the commissioners, along with other volunteers, lived among yellow fever patients in wards from which mosquitos were excluded, and remained free from infection even though surrounded by the excreta and vomit of these patients, proving that transference of the disease takes place normally through the bite of the mosquito alone. Dr. Agramonte was a native of Cuba and left the island at 2 years of age when his father was killed by Spanish troops. He qualified at Columbia College in New York in 1892, and soon after became bacteriologist to the Board of Health. In 1901 he was transferred to the University of Havana, leaving that post to be head of the department of tropical diseases at the new medical school of Louisiana State University. This summer he was elected president of the Pan-American Medical Congress to be held next year in New Orleans.

SPECIAL ARTICLES

STUDIES OF THE MECHANISM OF UPPER RESPIRATORY INFECTION.

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For many years investigators have sought to discover the aetiology of the common cold. Many organisms have at different times been assigned a causative rôle, and frequently the evidence in favour of one or another agent has been impressive. In general, however, such organisms have seemed to be of local importance, and not to have had a universal significance. Many of the organisms described are well-recognised pathogens of the upper respiratory tract, and their importance is apparent to all students of upper respiratory disease, but none has an established position as the principal initiating agent of upper respiratory infection. Our own studies of this type of infection have led us to believe that the visible bacteria that can be more or less readily cultivated from the upper respiratory tract during infection are not of primary significance, but must for the most part be assigned the rôle of secondary invaders.

Important evidence has already been accumulated suggesting that the primary agent of the simple type of respiratory infection that goes by the name of the common cold may belong to the order of the ultra-microscopic viruses.[1] The studies which we have carried out offer definite evidence in support of this point of view.

Observation has confirmed the opinion that anthropoid apes, particularly the chimpanzee, are susceptible to acute upper respiratory infections that resemble in every respect the same type of infection in man. Such infections are readily communicable from man to the ape, and from ape to ape. Study of the bacterial flora of the upper respiratory tract in chimpanzees shows it to resemble that of man to a remarkable degree. These animals, therefore, should prove to be excellent animals for study of the experimental transmission of acute upper respiratory infections. Such in fact has proved to be the case.

EXPERIMENTAL INFECTION IN APES.

A colony of young chimpanzees was assembled and placed under observation. To guard against accidental infection the animals were kept in specially organised rooms under rigid quarantine whenever experimental procedures were in progress. When such animals are inoculated intranasally with bacteria-free Seitz filtrates of nasopharyngeal washings from individuals with acute colds, within from 24 to 48 hours the apes begin to manifest signs of upper respiratory infection. These signs are clearly objective, and consist of sneezing, coughing, nasal obstruction, and a mucoid or muco-purulent nasal discharge. These phenomena persist for from five days to two weeks. Such experimental infections are readily communicable by contact to healthy chimpanzees placed in the same cage, and can be passed experimentally from animal to animal by the technique described. Experiments of this nature have been successfully performed over a period of several years and leave no doubt that the infection in question is communicable by means of a filtrable agent.

Perhaps the most significant change, from a bacteriological standpoint, has been the increase of activity on the part of the potential pathogens habitually present in the throat flora of the apes. Coincident with the appearance of symptoms, pneumococcus, *Streptococcus hæmolyticus*, *Bacillus pfeifferi* have developed in greatly increased numbers, and have spread over a wide area of the nasopharyngeal mucous membranes. These organisms become at this time conspicuous even in the nose where they are seldom or never present under normal conditions. We have also observed that during the period of acute infection animals who carry hæmolytic streptococcus may readily communicate this organism to contact animals. Such transference does not easily take place from carrier to contact in the absence of acute respiratory infection in the carrier animal.

EXPERIMENTAL INFECTION IN MAN.

Studies of experimental transmission of colds have also been carried out with human subjects. Human volunteers were carefully isolated and inoculated intranasally with bacteria-free filtrates from individuals suffering from acute colds. After an incubation period of about 24 hours the inoculated subject has manifested the signs of the common cold, and has run a typical course of symptoms lasting from one to two weeks. Such experimental colds in human beings have been passed in series from individual to individual. These human experimental colds differ bacteriologically from the experimental cold in the chimpanzee in that there is no immediate evidence of increased activity on the part of the potential pathogens harboured in the human nasopharynx. The same difference in bacterial activity is manifest between the spontaneous cold in the chimpanzee and the spontaneous cold in human beings.

THE VIRUS IN VITRO.

When we had satisfied ourselves that the common cold is caused by a filtrable virus, our next efforts were directed toward cultivating the causative agent in vitro. The first steps in the successful carrying out of such a procedure must naturally be directed toward an attempt to preserve the virus in an active form outside the animal body.

In order to test survival of the virus after removal from the human body, nasopharyngeal washings were obtained in the manner previously described from individuals within the first 24 hours of a typical attack of common cold. The material was quickly passed through a Seitz filter, and the filtrate was subsequently tested for its capacity to induce the symptoms of the common cold in chimpanzees and in human volunteers. The filtrates with cystein hydrochloride added were preserved anaerobically under vaseline seal both at room and at ice-box temperature. In a few instances the filtrate was concentrated to approximately one-seventh its original volume by vacuum distillation. Of 12 inoculations of filtrates preserved in this manner nine have produced in the inoculated subject typical symptoms of

the common cold. The duration of time of preservation of active filtrates has ranged from 4 to 13 days. Tests of survival beyond the thirteenth day have not been made. Positive results have been obtained with filtrates kept at room temperature, and with those kept at ice-box temperature, and with both the unconcentrated filtrate and with that concentrated by vacuum distillation. From these experiments we conclude that the virus of the common cold survives under suitable conditions for at least 13 days after removal from the human body.

In view of the capacity of this virus to survive in what might be considered an adverse environment an effort was next made to cultivate the agent in living tissue medium. Foster [2] has previously reported the cultivation of an organism from patients suffering from the common cold. The method of culture chosen by us has been that developed by Maitland and Maitland,[3] and by Li and Rivers.[4]

Ten-day chick embryos are hashed and suspended either in Tyrode's solution or in buffered bouillon prepared from a special peptone,* and to which 0·1 per cent. gelatin has been added. All solutions, both those used for nasopharyngeal washing and for cultivation, contain 1 to 2000 cystein hydrochloride. At all times an effort has been made to protect the agent against the deleterious action of peroxides. The cultures were incubated under vaseline seal at 37° C. for from four to nine days.

The material used for cultivation was obtained in the following manner: nasopharyngeal washings were made from a patient with a typical cold of more than moderate severity. The washings were passed through a Seitz filter and preserved at ice-box temperature under vaseline seal in the special peptone broth described. On the ninth day of preservation a volunteer was inoculated intranasally, and within 24 hours developed typical symptoms of the common cold. As soon as the signs of the disease were well marked, nasopharyngeal washings were prepared in the usual manner and preserved for five days at ice-box temperature. After this interval a second volunteer was inoculated, who also developed symptoms of a cold within the usual incubation period. Nasopharyngeal washings were again prepared, filtered through a Seitz filter, and concentrated by vacuum distillation. After ten days' preservation at ice-box temperature a third volunteer was inoculated. This individual developed a cold of unusual severity, possibly indicating an accession of virulence by the virus. Nasopharyngeal washings were prepared from this individual after the usual interval, filtered through a Seitz filter, concentrated seven times by vacuum distillation, and preserved for five days at ice-box temperature. After the lapse of this interval 0·25 c.cm. of the concentrated material was inoculated into tissue medium of the nature described. The culture was incubated for five days at 37° C. At the end of this time a human volunteer was inoculated and within the usual incubation period developed a cold with symptoms of rather a mild character.

From this time on the material was carried in tissue medium and transferred at intervals of from three to nine days. We believe the preferable interval between transfers to be four days. From time to time cultures were chosen for the inoculation of experimental subjects. The third culture representing a dilution of the original material of approximately 1–2000 was inoculated into a chimpanzee. This animal developed symptoms of a cold after an incubation period of four and a half days. An uninoculated companion animal kept in the same cage developed symptoms of a cold at the same time. The sixth culture, representing a dilution of the original material of approximately 1–2,000,000, was inoculated into three human volunteers. Of these, two developed after 24 hours typical colds of moderate severity. The tenth culture, representing a dilution of the original material of approximately 1–20,000,000,000, was inoculated into three human volunteers. Of these, one developed such mild symptoms of respiratory infection as to be considered doubtful. The twelfth culture, representing a dilution of approximately 1–2 trillion, was inoculated into three human volunteers, and of these one developed a cold of mild symptoms. The fifteenth culture, representing a dilution of approximately 1–2 quadrillion, was inoculated into three human volunteers. Of these, two developed colds of unusually severe symptoms, complete nasal obstruction, frontal headache, malaise, loss of appetite, nasal discharge, and pronounced cough. Nasopharyngeal washings were prepared from these two individuals, filtered through a Seitz filter, and concentrated by vacuum distillation. Two human volunteers were inoculated after a short interval with these concentrated filtrates and within 24 hours both developed colds, one with mild symptoms and the other with the typical features of a head cold.

* Especially prepared peptone obtained through the kindness of Dr. René Dubos, of the Hospital of the Rockefeller Institute.

DISCUSSION.

The evidence presented by these experiments indicates that the virus of the common cold survives for a considerable length of time after removal from the human nasopharynx, and that the agent reproduces itself in vitro when cultivated in suitable tissue medium. The total duration of life of the culture virus from the time of removal from the human subject has been 74 days. The final dilution of the original material is so great as to rule out conclusively the presence of any significant amount of the original virus. Controls of the constituents of the medium and of the tissue medium have been negative. In one instance when a volunteer received active culture heated for half an hour at 80° C. there appeared symptoms of upper respiratory irritation which lasted for less than 24 hours.

From these studies the conclusion may be drawn that one type of acute upper respiratory infection is caused by a filtrable virus. In addition to initiating the symptoms of infection this agent may be considered to provoke increased activity of any potential pathogens that may happen to be present in the respiratory tract, and, furthermore, to render the respiratory tract susceptible to implantation by pathogenic organisms from the exterior. An interesting observation in connexion with this point of view is that in individuals stigmatised with rheumatic disease many times a recrudescence of the rheumatic process is occasioned by an attack of the common cold. From the above observations the inference may be drawn that the virus of the common cold in the individual plays the double rôle of initiator and promoter of upper respiratory infection.

The significance of this virus in the ætiology and epidemiology of respiratory infection of the community as a whole is still obscure, but certain interesting facts indicate its great importance. Studies [5] of outbreaks of common cold in representative population groups show a high morbidity-rate for this infection of cyclic character. The peaks of incidence are three in number, and occur in the early fall months, September and October, the mid-winter months, January and February, and the spring months, April and May. The character of the different

outbreaks varies in severity, and there seems to be a progressive increase in the seriousness of the secondary infections of the respiratory tract as the winter advances into spring. For example, the amount of absenteeism from work is much greater during the January outbreak than during the October outbreak. The incidence of the pneumonias is higher during the former period than during the latter. Furthermore, since the pandemic of influenza in 1918, all outbreaks of influenza in New York City have occurred in the early months of the year, thus coinciding in time with one of the periods of maximum incidence of the common cold. Studies conducted by us have shown, considering the respiratory year, so to speak, as running from September to June, that as this year advances there is an increasingly wide distribution in the nasopharynx of healthy individuals of such potential pathogens of the upper respiratory tract as pneumococcus, *B. pfeifferi*, and *S. hæmolyticus*.

The interpretation of these observations is not yet clear, but it seems not unlikely that as a consequence of the repeated cycles of common cold throughout the respiratory year an increasing opportunity is afforded the potential and sometimes secondary pathogens of the respiratory tract to cause infection. As a result of this their distribution is promoted and their virulence perhaps enhanced. This type of associated activity makes it seem not unlikely that certain outbreaks of so-called influenza or grippe may result from the coincidence of an epidemic outbreak of the common cold, with a period of wide distribution and increased virulence of the well-recognised pathogenic organisms of the upper respiratory tract. However this may be, there is no doubt that the virus of the common cold plays a most important rôle in promoting respiratory infection in the community as a whole, in addition to its obvious significance in respiratory infection in the individual.

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PARIS.

(FROM OUR OWN CORRESPONDENT.)

TROPICAL MEDICINE AT THE COLONIAL EXHIBITION.

THE Journées Médicales Coloniales at the Colonia Exhibition at the end of last month (July 22nd–31st constituted a veritable international conference on tropical medicine. It opened in the great hall of the Cité des Informations under the chairmanship of Marshal Lyautey, who paid this tribute to medicine —that in many cases it had been able to replace battalions in the pacification of new countries. Prof. Tanon outlined the programme of the conference, which he hoped would prove a milestone in the evolution of tropical hygiene. The number and standing of the official delegates from Great Britain and her dependencies showed how great was the importance attached to it in this quarter.

Among the numerous and most interesting reports presented was one by Dr. A. Thiroux on birth-rate and infant mortality in the French colonies. It was based on the study by Colonel Cazanove, presented in 1930 to the Office International d'Hygiène Publique. The birth-rates showed remarkable local differences, ranging as they did from 7 to 84 per 1000, the last-named prodigy of fecundity being claimed by a district in the Cameroons. The infant mortalities also showed wide fluctuations, ranging from 155 to 319 deaths in the first year of life for every 1000 live births. He headed the list of the causes of death in infancy with malaria, giving as an example the findings in Antananarivo of malarial enlargement of the spleen in 77 per cent. of the school-children. In a paper on sleeping sickness in French equatorial Africa, Dr. A. Sicé stated that among 830,448 natives inspected in 1929 the morbidity-rate from this disease was about 4 per cent. After enumerating a list of measures and remedies, he concluded that trypanosomiasis has ceased to be a danger for the economic future of French equatorial Africa. Other speakers dealing with trypanosomiasis in French occidental Africa stated that on the whole, and since 1920, this disease has waned, although here and there it has gained new ground. The reports on the prevention and treatment of leprosy showed how great have been the changes of late years in the attitude of medical science towards it. Dr. Stevenel discussed recent experiences with chaulmoogra oil, the only drug which, in his opinion, has hitherto proved indubitably effective, and with regard to prophylactic measures he insisted that they should not be more cruel than those now recommended in tuberculosis. Dr. Thomas Cochrane, discussing the prophylaxis of leprosy in the British African Colonies, also showed how impossible it would be, physically and financially, to apply measures of strict isolation to the five to ten million lepers supposed to be living in the world to-day.

A CONGRESS OF DENTISTS.

The eighth International Dental Congress, which opened on August 2nd at Versailles, under the patronage of the President of the Republic, was held during the following days in the Grand-Palais in Paris. More than 3000 persons took part, and 33 governments sent official representatives. The President, Dr. G. Villain, in his opening address, traced the history of dentistry since the congress last sat in Philadelphia in 1926. Up till then dentistry had been overshadowed by a vogue which traced an infinite number of evils to the teeth themselves, the medical profession in many countries taking such a wholesale and severe attitude towards the alleged sins of the teeth that their extraction had become the most habitual therapeutic act, and the dentist had found himself "demoted" to the elementary rôle of tooth extractor. Dr. Villain considered that the fortunate passing of this crisis had led, in the course of the last five years, to the development of research into prosthetic restoration. Another important development in dentistry during the last five years was, in his opinion, the work of Mellanby and others on the relation of caries to metabolism and diet.

THE LICENSED BROTHEL.

It is pretty safe to prophesy that if and when the official regulation of prostitution is discontinued in France this change will be due not so much to the experience of other countries as to the tentative experiments now being made here and there in France itself. The abolition of the controlled brothel in Grenoble is being followed with the keenest interest by the abolitionists and their opponents, who keep

up an interesting correspondence in the *Siècle Médical*. Meanwhile the town of Nancy has decided to follow suit, and its mayor has issued an order closing all the *maisons de tolérance*, whose licences are to be withdrawn on Nov. 1st.

UNITED STATES OF AMERICA.

(FROM AN OCCASIONAL CORRESPONDENT.)

POLIOMYELITIS.

SOMETHING like an epidemic of poliomyelitis has made its appearance in certain of the Middle Atlantic States and shows a tendency to spread westward. The increased incidence was first noted in New York City during the week ending July 25th, in which 195 cases were reported, as compared with 35 cases for the first six months of the year. A sharp rise in incidence was also reported at the end of July by the Connecticut State Department of Health. During the first half of August the incidence of poliomyelitis in the state of Illinois has been three times the expectancy. Although this does not represent a rise comparable to that in the east it shows a tendency for the disease to spread along the line of travel.

Various State health departments have taken measures to meet the threatened attack. Connecticut, New York, and Illinois are providing consultant diagnostic service and immune serum. In Connecticut serum has been obtained from the Newington Home for Crippled Children. In New York State appeals for blood from those who have had the disease have been broadcast, and Governor Roosevelt has set an example by giving, for the second time, some of his own blood. Hospitals are advised to be prepared to take cases under conditions of isolation and to provide themselves if possible with a Drinker respirator for the treatment of cases with bulbar paralysis.

A conference of New England health officials was held in Boston on August 3rd. At its close a statement was issued warning parents that "Perhaps the most suggestive early sign warranting attention is a stiffness of the spine and neck in a sick child, best shown when the child is asked to sit up in bed and bend the head forward between the knees. Infantile paralysis," it is stated, "is spread by personal contact with patients or healthy persons carrying the infection. Children's contacts therefore should be restricted to the usual groups of associates. Camps and children's institutions are advised to discourage visiting by outsiders. Children now in camps under controlled conditions and observation had better stay there rather than increase their contacts by leaving." Even as far west as New Mexico warning circulars have been sent to the medical profession advising of the chief diagnostic symptoms of poliomyelitis in the prodromal stage and of the significant laboratory findings in these early cases.

DEATH OF LEE KAUFER FRANKEL.

By the death in Paris on July 25th of Lee K. Frankel, Ph.D., America loses one of its outstanding leaders in public health. He was for nine years manager of the United Hebrew Charities of New York, and then for a short time special investigator for the Russell Sage Foundation. But it was in 1909, when he joined the staff of the Metropolitan Life Insurance Company and organised its welfare division, that the nation as a whole began to feel the impress of his influence. The work which he organised, both in educational literature and particularly in the public health nursing service, was quite original as a contribution of insurance practice and extremely influential in promoting public health. Dr. Frankel was vice-president of the National Tuberculosis Association in 1914, president of the American Public Health Association 1919, and of the National Health Council, child of his own organising genius, from 1923 to 1926. The Framingham tuberculosis demonstration was another invention of his fertile mind. Numerous public health organisations benefited from his advice. At the time of his death he had recently completed a survey of social insurance in Europe.

IRELAND.

(FROM OUR OWN CORRESPONDENT.)

POOR-LAW SALARIES IN COUNTY WEXFORD.

THE County Wexford board of health has been informed by the Department of Local Government and Public Health that owing to the continued neglect of the board to fix a suitable scale of salaries for the dispensary medical officers of the county, the Minister felt constrained to issue an order fixing the salaries of these officers. The salaries were now determined to be on a scale beginning at £200 a year, and increasing by £5 a year to £300 a year, to be applied retrospectively. The order was not to affect any existing officer adversely. The sealed order was enclosed. The order affects eight medical officers, and brings about a total increase of £390 a year. With a scale of salaries established in Wexford, there is now a scale in force in every county in the Irish Free State. There are, however, considerable differences between the scales in different counties.

THE DERBY SWEEPSTAKE.

The Irish Hospitals Trust published last week a financial statement concerning the sweepstake on the Derby. The total receipts, after deducting £38,000 for sellers' prizes, came to £2,789,696. Of this sum £697,424, or 25 per cent., was distributed to the hospitals; £1,902,500, or 68·2 per cent., was spent in prizes; and £189,772, or 6·1 per cent., went in expenses. Of the latter sum, £58,266 was paid to Hospitals Trusts, Ltd., as promoters of the sweepstake. The grants to hospitals were divided among 34 institutions. The highest grant was £48,819, or 7 per cent. of the total, which was given to the Royal City of Dublin Hospital. Five hospitals—Sir Patrick Dun's, Meath, Richmond, Cork Street, Coombe—got 6 per cent., or £41,845 each. Various grants were given to the others, the smallest grants, 0·5 per cent., or £3487, reaching the Martin Hospital, Portlaw, and the Drogheda Memorial Hospital, The Curragh. Preparations are far advanced for the sweepstake on the Manchester November Handicap. The list of participating hospitals has not yet been compiled.

THE METROPOLITAN HOSPITAL.—The annual report of this institution (Kingsland-road, E.) states that serious defects in the fabric have been discovered. The fire staircase must be renewed at once, and fractures in the main structure, necessitating repairs of considerable magnitude, must be put in hand shortly. The cost of these works will be between £6000 to £7000, and an effort is to be made to raise this sum before launching the general appeal that is in contemplation. When that appeal is made over £50,000 will be asked for to pay off the debt of £18,000 on the nurses' home and to meet the cost of essential improvements.

PANEL AND CONTRACT PRACTICE

ETHICS OF CONSULTATION.

AT the annual panel conference in October last attention was given to smooth working of additional benefits under the N.H.I. Acts, and particularly to the relation of panel practitioner to consultant in his various guises. It seemed to the representative of East Sussex for instance that his patients were apt to disappear into sanatoriums, rheumatic clinics, and other forms of special treatment, and return to him later without any information on treatment received or advice given. He thought, and the conference agreed with him, that the accepted ethics of consultation should apply to all these situations in insurance practice. The matter was referred to the central ethical committee of the British Medical Association which decided that the rules drawn up for private practice applied equally well to insurance practice if read in conjunction with the rules guiding the practitioner when called upon by a third party to make an examination. The two sets of rules which are of fundamental importance appear as Appendix C to the report of the Insurance Acts Committee printed in the Supplement to the *British Medical Journal* for August 29th. In these rules the term "consultant" is to be understood as meaning any practitioner who is called upon by the patient or by any person acting on behalf of the patient, to advise in special circumstances with regard to a patient who is already under the care of another practitioner, that other being referred to as the "attending practitioner."

WHERE CONSULTATION IS REQUIRED.

In the interests alike of the public and of the medical profession, it is the duty of a medical practitioner to accept the opportunity of consultation, especially upon obscure and difficult cases, or where the patient or his friends desire it.

In the following cases it is especially the duty of the practitioner in attendance to endeavour, if practicable, to obtain the assistance of another opinion:—

(*a*) When a question arises of the propriety of performing an operation or adopting some course of treatment which may be dangerous to life or permanently injure the condition of the patient, especially if the condition which it is thought to relieve by this treatment be not itself dangerous to life.

(*b*) When a question arises of destruction of a fœtus, or unborn child, in the interest of the mother, especially if she is not in labour.

(*c*) When the practitioner in attendance is in doubt either as to the diagnosis or as to the treatment to be followed, and delay in arriving at a decision might be dangerous.

(*d*) When there is evidence of anxiety on the part of the patient or his friends as to the correctness of a diagnosis or of the treatment pursued.

(*e*) When the attending practitioner has reason to suspect:—

(i) Performance of an illegal operation.
(ii) Administration of poison.
(iii) Commission of any other criminal offence.

CHOICE OF CONSULTANT.

It is expedient in the interests of the patient that the choice of consultant should usually be left to the practitioner in attendance. Nevertheless, a practitioner is not justified in refusing to meet a consultant selected by the patient, or the patient's friends, unless he is satisfied that the proposed consultant is not qualified by knowledge or special experience to advise upon the case, or that he is one of whom he is debarred from meeting. It is the duty of a practitioner to refuse to meet in consultation:—

(*a*) An unregistered person.

(*b*) A practitioner whose exclusive profession of any peculiar system of treatment would render consultation futile.

(*c*) Any practitioner whose conduct has, after due inquiry, been declared by some recognised body of the medical profession to be detrimental to the honour and interests of the profession, such declaration not having been made inoperative by any subsequent pronouncement of the body in question or of any superior ethical tribunal.

PROCEDURE AND ETIQUETTE.

The arrangements for consultation should be made by the attending practitioner. If a practitioner receives an application to act as consultant from any person other than the attending practitioner of the patient, he shall not see the patient without the consent of the attending practitioner. The following rules should be observed, unless there is substantial reason in any particular case for departing therefrom:—

(*a*) If the consultation is not held at the patient's house, it generally takes place at the house of the practitioner consulted, who also fixes the hour of meeting, unless otherwise amicably arranged.

(*b*) All parties to a consultation should be punctual. If the attending practitioner does not keep the appointment, the other or others may, after a reasonable time, see the patient, and leave his, or their, conclusions in writing, in a closed envelope addressed to the attending practitioner.

(*c*) Before seeing the patient, the attending practitioner should, as a rule, give the consultant a brief history of the case.

(*d*) On entering the room of the patient, the attending practitioner should precede the consultant, and should, if necessary, introduce him to the patient, and the attending practitioner should be the last to leave the room. The diagnosis, prognosis, and treatment should be discussed in private.

(*e*) The opinion on a case, and the treatment, should be communicated to the patient or the patient's friends by the medical man consulted, in the presence of the attending practitioner.

(*f*) If it is found necessary that the patient or his friends should be made aware of a difference of opinion among the practitioners taking part in a consultation, it is the duty of the consultant, jointly with the attending practitioner, to communicate this information.

(*g*) The practitioner in attendance should ascertain previously, and inform the patient or his friends, as to the amount of the fee, which should be paid to the consultant at the time.

(*h*) If for some reason a personal consultation is impossible, the attending practitioner should write a letter introducing the case, and should courteously and punctually reply to any communication from the consultant, and the latter should write and forward his opinion, along with any prescription he may advise, in a closed letter addressed to the attending practitioner.

(*i*) Arrangements for a future consultation should be left to the initiative of the practitioner in attendance.

RELATION OF CONSULTANT TO PATIENT.

Great care should be exercised in making any observations in the presence of the patient on the nature of the malady, its probable issue, or treatment pursued, and all criticisms or reflections on the practitioner in attendance must be avoided. Differences of opinion, so long as there is final agreement among the parties to the consultation, are not to be revealed, but if agreement as to diagnosis and treatment should not be possible and the consultant is convinced that

the future well-being of the patient is concerned in his so doing, he should inform the patient in the manner prescribed above.

It is the duty of the practitioner in attendance loyally to carry out the measures agreed upon at the consultation, and he should refrain from making any radical alteration in such measures except upon urgent grounds or after adequate trial.

In recognition of the position of trust in which he is placed towards the attending practitioner, the consultant must not, by unduly ingratiating himself or otherwise, attempt to secure for himself a patient, or the relative of a patient, whom he has seen in consultation, and must exercise scrupulous care to avoid disturbing the confidence of the patient in the attending practitioner.

If the consultant wishes to make any communication with regard to any case which he has seen in consultation, he should only do so through the attending practitioner, and should not discuss the case with the patient or any of the patient's friends, in the absence, or without the consent of, the attending practitioner.

A practitioner who has seen a case in consultation should not supersede the attending practitioner during the illness with regard to which the consultation is held, and, if he be asked to attend or prescribe in any future illness he should only do so after explanation with the attending practitioner, unless circumstances make this impracticable.

Third Party Consultations.

There remains to consider the special case of patients under care of other practitioners. Rules in respect of position of medical practitioners called upon to examine, otherwise than by request of patient or persons acting on patient's behalf, are as follow. They do not apply to certifying factory surgeons.

NOTICE OF VISIT.

The medical inspector should give the medical attendant such notice of the date, time, and purpose of his visit as will afford reasonable opportunity for the medical attendant to be present should he or the patient so desire. Exceptions are :—

(*a*) When circumstances justify a surprise visit.

(*b*) When circumstances necessitate a visit within a period which does not afford time for notification.

(*c*) When the medical inspector, after due inquiry made, has no information as to whether the patient is under medical care.

Where the medical inspector has availed himself of any of the above exceptions, it shall be his duty to inform the medical attendant, if any, of the fact of his visit, and the reasons for his action.

THE VISIT ITSELF.

The medical attendant must not put any unnecessary difficulties in the way of fixing a time convenient to both practitioners. If the medical attendant fails to appear at the time stated, the medical inspector may proceed with his examination forthwith. The medical inspector must not, without the consent of the medical attendant, do anything in the course of his examination which involves active interference with the treatment of the case. Where the medical attendant fails to communicate with the medical inspector, the medical inspector shall at his discretion and subject to the consent of the patient, make any examination he may consider necessary.

THE REPORT.

The medical inspector must not make any comments to the patient which are of the nature of criticisms of, or reflections upon, the treatment, nor must he express, without the concurrence of the medical attendant, any opinion to the patient as to the ætiology, diagnosis, or prognosis of the case. His duty is strictly confined to examining into such matters as are necessary for the purposes of his report, and reporting to his employer, and to his employer only, his conclusions from such examination.

If the medical inspector finds it necessary to report to his employer that any modification in the treatment which is being carried out is, in his opinion, necessary to the more rapid recovery of the case, he shall, if possible in the first instance, so inform the medical attendant.

MEDICINE AND THE LAW.

Professional Registration—The Architects' Success.

STATUTORY registration, a principle of proved value for establishing professional standards in the public interest, has now been applied to architects. The Medical Act, rather than the Dentists Act, is the precedent which has been followed; that is to say, there will be a monopoly of the title of "registered architect," but no monopoly of practice; Parliament, therefore, has not needed to define the practice of architecture. On the other hand the Dentists Act, rather than the Medical Act, is followed in the provision which allows a right of appeal to the High Court to a person aggrieved by a decision to remove his name from the register or by temporary disqualification. Among other parallels or variations we may note that an architect's name may be removed from the register on conviction of a criminal offence or for "conduct disgraceful to him in his capacity as an architect." The traditional phrase "infamous conduct," sometimes a source of popular misunderstanding in relation to medical practitioners and dentists, has thus been dropped, and the language of the Veterinary Surgeons Act has been adopted instead. Such conduct in an architect will be referred to a disciplinary committee, which is assured of a legal element in its quasi-judicial functions because, in addition to members appointed by the Office of Works and the Ministry of Health, there are to be two members nominated by the President of the Law Society. The qualifications for registration include anyone who applies within two years, and satisfies the Architects' Registration Council that at the commencement of the Act—i.e., Jan. 1st next—he is or has been practising as an architect in the United Kingdom. There will thus be a class of "1931 architects," corresponding to the "1921 dentists."

The architects' profession is to be congratulated on obtaining this degree of statutory regulation. The establishment of a single central supervising body is valuable in itself. Much may be done even by a system of mere voluntary registration such as has been initiated lately, for instance, for biophysical assistants in the hope of ridding the public and the medical profession of quackery in certain physical treatments. Statutory sanction gives, of course, a national recognition as well as supporting the system of registration by the infliction of penalties for its abuse or infringement.

MOORFIELDS EYE HOSPITAL.—The annual report of the Royal London Ophthalmic Hospital, Moorfields, states that last year 2547 in-patients and 52,372 out-patients were treated, and that the income was £34,279 and the expenditure £32,480. The out-patients' department urgently needs enlargement; the cost will be £40,000, towards which £13,000 is available.

CORRESPONDENCE

IMPRISONMENT FOR MENTAL DISORDER.

To the Editor of THE LANCET.

SIR,—The letters of Prof. Millais Culpin (p. 373) and of Dr. Russell Brain (p. 426) raise an issue in which the National Council for Mental Hygiene is especially interested. The Council has in the course of the last few years collected records of a not inconsiderable number of cases in which the magistrates have refused to take into consideration the evidence of doctors as to abnormality in the mental condition of offenders, or have refused to permit examination by medical experts where this has been asked for in the interests of the offender. In addition to these there have been cases where the existence of an abnormality was undoubted and in which the magistrate has passed sentence without taking into consideration the medical aspect of the case. Three instances may be cited:

(1) A man of middle age had repeatedly written letters of an abusive nature to the municipal authorities accusing them of appropriating land which formed a part of his property. Medical evidence was adduced to show that the man was suffering from delusions of persecution. The magistrate sentenced the man to a term of imprisonment with the comment that "It was the function of the Court to cure delusions."

(2) A boy of about 17 years of age, described as of a moody and seclusive temperament and who had on several occasions previously behaved in a sufficiently bizarre manner to cause comment, set fire to some ricks. No motive was found and the boy was unable to give any reasonable explanation for his act. No investigation of the boy's mental state was made and he was duly sentenced.

(3) An educated woman of early middle age, a school teacher with an excellent record, was caught stealing some small articles in a store. No investigation was made into her mental condition although it was clearly established that she had no real need of the articles and could easily have paid for them. In passing sentence of imprisonment the magistrate said that although she was a woman of education and means she had yet behaved like a common thief, a fact which he considered an aggravation of the offence.

Comment on these cases is superfluous. On the other side of the picture and to show the value and importance of obtaining expert opinion the following cases which passed through my own hands may be cited:

(1) A married woman of early middle age, whose husband was of the respectable working class, was charged with writing abusive letters to certain public men making offensive allegations. Her previous record was blameless. The magistrate referred her for investigation to be made into her mental condition. It appeared that she was now at the menopause, and that for the last two years had been showing emotional instability and character changes during her periods, at times becoming definitely deluded, with ideas of persecution. She undertook to attend the hospital regularly for treatment and the case was dismissed. Needless to say there has been no recurrence of the offence.

(2) A boy, aged about 18, was charged with attacking a girl in a quiet street and trying to snatch her handbag. The boy's father, on his own initiative, brought him up to the hospital clinic before the trial for investigation into his mental condition. The boy had a perfectly satisfactory record at school and at home. He was, however, rather afraid of his parents and was latterly showing evidence of impaired memory and undue shyness and nervousness. His employment was uncongenial, and he was told to go and interview a new employer. Afraid to do this he loitered about, and forgetting where he had left his bicycle wandered out of the town. Towards evening he found he had no money and was some distance from home. He ascribed snatching the handbag to an unreasoning impulse. He finally returned home in a dazed condition and in great mental distress at what he had done. An uncle now offered the boy a post in the country. The girl did not wish to prosecute. A letter was written to the magistrates putting the medical factors of the case before them. The case was dismissed and the boy is now in the country and doing well.

At the biennial conference of the National Council for Mental Hygiene, held in May last, a general demand made itself felt for increased facilities for the education of magistrates on the subject of mental hygiene. The Council in its lecture courses held twice yearly always includes some aspect of this problem. The Tavistock Square Clinic for Functional Diseases has given lectures for magistrates and the National Council is arranging further lectures in the immediate future. One of the difficulties, however, is to procure the audience. Many magistrates, are busy people, and for those in the provinces especially it is difficult to afford adequate facilities, even if they were prepared to make use of them. The new Mental Treatment Act and the increase of out-patient clinics for the treatment and investigation of cases of emotional instability and early mental disorder should, however, in course of time provide a place to which doubtful cases can be referred in every area. It is important to educate the public as well as the magistrate up to the use of these invaluable facilities.

I am, Sir, yours faithfully,

DORIS M. ODLUM,
Hon. Sec. for Propaganda, National Council for Mental Hygiene.

August 23rd, 1931.

TUBERCULOSIS IN INFANTS.

To the Editor of THE LANCET.

SIR,—In your annotation of August 15th (p. 363) on Tuberculosis in Russian Infants, attention is drawn to the early appearance of this disease. It is often assumed in England that if a baby is breast-fed by an apparently healthy mother, tuberculosis need not be feared. Two cases recently encountered show that this may be a very erroneous idea, and that tuberculosis may be met in infants where there is no suggestion of the infection in either parent, or either family, nor any evidence of contact during the few weeks of life with any affected person. The conclusion to be drawn is that in some cases, even with no family history of tuberculosis, the susceptibility is very high, and massive infection is therefore unnecessary.

An entirely breast-fed infant, aged 43 days, weighing 7 lb. 1 oz. was admitted to Booth Hall Hospital for Children as a weakly infant with a history of having on the day of admission suddenly commenced to droop, the child having been perfectly well until then. Birth weight was 6 lb. 2 oz. When five weeks old the weight was 7 lb. 6 oz. and two days before admission 7 lb. 5 oz. The mother was admitted to feed the infant. There was slight vomiting on arrival, but no more until once 11 days later. There was no diarrhœa. A week later the child appeared hungry and lively, test feeds showing that he was obtaining about 5 oz.—a considerable amount for a seven weeks' baby. The next day one feed was refused, the general condition being poor. On succeeding days the temperature rose to 99° F., to 100°, and to 101°, dropping abruptly on the next day. X ray of the chest showed definite parenchymatous infiltration of both lungs, and the child was considered to be suffering from broncho-pneumonia. On the day the temperature fell the child had to be fed from a pipette with milk taken from the breast, and about 10 o'clock died quite suddenly. The Wassermann test was negative. Examination post mortem showed generalised miliary tuberculosis involving lungs, spleen, and liver, but not the brain.

In this case there was no direct evidence of parental infection; but the mother, during her pregnancy, drank unboiled milk from a source which is frequently found to be tuberculous. The mother was requested

to return so that a sample of her breast milk might be examined for tubercle bacilli, but she failed to put in an appearance. Thanks to the kindness of Dr. D. P. Sutherland, senior tuberculosis officer for the City of Manchester, and Dr. Norah Smith who is in charge of the maternity and child welfare department, an extremely complete investigation was made into the previous history of this case as regards possible contact with a known case of tuberculosis, or the ingestion of any contaminated milk by the patient, with negative results.

Whilst interest was still aroused by this case, a similar one, as so frequently happens, came to my notice.

A child, aged 8 months, still on the breast, was admitted on account of gastritis and constipation. The combination is not usual, and progress was not satisfactory, there being continuous loss of weight. Vomiting and constipation persisted, and on the eleventh day after admission there was rigidity of the neck muscles, and an occasional squint, a note being made "the child does not appear dyspeptic; looks more like cerebral tumour." Examination of the eyes next day showed slight blurring of the nasal side of both discs, the vessels being normal. Lumbar puncture gave 20 c.cm., of clear colourless fluid under pressure, with no improvement in the condition. Examination of the cerebro-spinal fluid showed that the cells were increased to 29 lymphocytes per c.mm., the protein being 120 mg. per 100 c.cm., but no tubercle bacilli were found. Progress was now much more definitely suggestive of meningitis, and further lumbar puncture gave 65 lymphocytes per c.mm., protein 200 mg. per 100 c.cm., and tubercle bacilli were found. The child died on the nineteenth day, examination post mortem showing one caseous lobule and small discrete tubercular nodules in the lower lobe of the left lung, and hilar and bronchial glands enlarged, caseous, and softened. The brain showed evidence of tuberculous meningitis.

Investigation of the previous history of this case by Drs. Sutherland and Smith showed that the child had been breast-fed, but that after six months old it occasionally had crusts dipped in unboiled milk. This milk-supply has been found to contain tubercle bacilli several times, but on no occasion this year. The mother stated that she was in the habit of taking the child to the "pictures." The baby also had teething powders mixed with milk, but the child had not been in contact with any known case of tuberculosis, nor are the parents considered tuberculous. Here again though there was no family susceptibility, and though the infant was continually breast-fed, very slight exposure to infection was sufficient to give rise to a pulmonary infection which probably was the origin of the meningitis.

I am, Sir, yours faithfully,

J. D. EWART, M.B. Lond.,
Medical Superintendent, Booth Hall Hospital for Children, Manchester, E.

August 25th, 1931.

TOURNIQUET PARALYSIS.

To the Editor of THE LANCET.

SIR,—I wish to associate myself with the views expressed by Mr. N. L. Eckhoff in your issue of August 15th (p. 343). The advantages of a bloodless field are, as he says, so many, it is unnecessary to stress them. It is in the manner of procuring this that the difficulty lies. I have long realised that when I apply a tourniquet (a flat Martin's bandage) I am risking a paralysis, and recently, wherever possible, I have used a pneumatic tourniquet; but up to the present I have not been able to find a really satisfactory one. It slips off, or the valve leaks, or it takes up so much room that it encroaches on the field of operation; in some cases it is not long enough to encircle the limb, and then I have to discard it and continue the operation without, or disarrange everything while the Martin's bandage is applied.

There is not as a rule much difficulty with the upper limb; it is the lower limb which causes most of the trouble. Where the thigh is fat and short, and the operation is on the knee-joint, it is quite impossible to obtain a bloodless field with the pneumatic tourniquet in use—which is a canvas-covered affair, part of a sphygmometer outfit—and one is forced to use the Martin. But the efficient application of even this may be difficult under the circumstances described. Unfortunately my experience with regard to the lower limb has differed from that of Mr. Eckhoff, for I have had a case of paralysis of the peroneal nerve following the application of the "old thick rubber tube" just above the knee. I had to perform an open osteotomy of the upper third of the right tibia for a lady aged 29 who had severe bow legs. Two days later complete peroneal paralysis was discovered. As this did not clear up, I explored the nerve at the site of operation to see if it had been injured, but it was unhurt. The paralysis did not resolve completely for over six months.

One other point. I do not allow any one but myself to apply the tourniquet—keen and enthusiastic residents are apt to apply it too tightly.

I am, Sir, yours faithfully,

PAUL BERNARD ROTH.
Welbeck-street, W., August 24th, 1931.

A REVOLVING SOLARIUM.

To the Editor of THE LANCET.

SIR,—The accompanying illustration (Fig. 1) shows a revolving solarium at Aix-les-Bains, France, which is, I am told, the only one of its kind in the world. The designer is Dr. Jean Saidman, director of the Institute of Actinotherapy in Paris, and I am indebted to him for kindly taking me over it.

Situated on a high hill amongst the mountains of Savoy, the solarium is visible a long way off and looks like an enormous aeroplane. On entering the door it seems incredible that such a network of machinery could have been constructed in so small a space. The ground floor consists of a miniature reception room, where the patients are indexed, and the solarium is kept in touch by telephone with the world below. In the middle of the body or tower is the lift, which brings one to the floor where the sun treatment is given. Apart from the reception office are the room of the director, where details of the patients' cases are filed, and here all the treatments are worked out and discussed by him and his able assistants, Dr. Robert and the electrotherapeutist, Mr. Sous. On the ground floor is also a waiting-room, which, like the other rooms, has silver wallpaper, glass doors, and silver chairs—everything to ensure light and brightness.

Proceeding to the top floor one has the sensation of being in the control tower of an aerodrome. In the middle of this floor is a small lounge, where the patients wait until they have their treatment; on the other side of that landing is the real heart of the whole mechanism, where the movements of the solarium are controlled so as to obtain the maximum intensity of the sun. The rays are measured by a special instrument, and the solarium is easily turned on its axis by an electromotor.

Dr. Saidman's method is based on three main principles, as follows: (1) The variations of the intensity of the constituents of sunlight (ultra-violet,

visible, and infra-red) should be taken into account. (2) In ordinary sunlight treatment the patient lies flat on a couch and receives the rays obliquely; whereas in the solarium he is so placed that the rays fall perpendicularly on his body, and this immensely increases the amount of energy received from the rays. (3) Usually, sunlight treatment is prescribed without any previous measurement of the patient's tolerance; but here a test for the sensibility of the skin is done as a precaution.

The treatment is given in the wings, which are composed of five rooms each. Each room or cabin, as shown in Fig. 2, has a couch which is tilted obliquely for the reasons already indicated. The sunrays are reflected on a special glass which concentrates them upon the patient. In one special cabin, however, it is possible to shut out all ordinary light and only ultra-violet rays are used.

While having treatment one has a magnificent view of the mountains, which naturally improves the therapeutic condition. Afterwards a cold douche is given, or a rub-down with eau-de-Cologne. Each cabin is in touch with the control room by telephone, and each amount of irradiation given to the individual patient shown on special meters. There is also a clock in each cabin, so that the time occupied in treatment is obvious at a glance. The risk of overdosage is much minimised by the preliminary "skin sensibility test." On days when Nature does not supply sufficient sunlight, use is made of artificial sunlight by means of mercury-vapour lamps, of special pattern designed by the director of the solarium.

Mostly during the treatment the patients drink the waters of the Source Saint Simon, which is one of the principal waters of Aix, and rises far away in the mountains. It is an alkaline water with diuretic action.

One floor lower than the cabins there is a very elaborate X ray plant generating 300,000 volts, which is used for deep X ray therapy; it has a very favourable effect on rheumatic affections of the joints. The patient is not in the same room as the X ray tubes, so there is no possibility of receiving electric shocks, and he is not made anxious. In order to achieve this he is placed on a special couch in the room below (on the ground floor) and his couch is pulled up to the ceiling. The rays are conducted to an opening in the ceiling, and the only thing the patient notices is the noise of the X ray plant above, which is not very great.

The solarium allows of all the applications of heliotherapy, but is primarily intended for the treatment of certain forms of rheumatism and arthritis. Neuralgias, cellulitis, and myositis are also benefited. The rays often reduce subcutaneous infiltrations and overcome muscular contracture. Synovitis and arthritis with fluid on the knees are also successfully treated in the solarium, and also old fractures, osteomyelitis, and recurring staphylococcal infections.

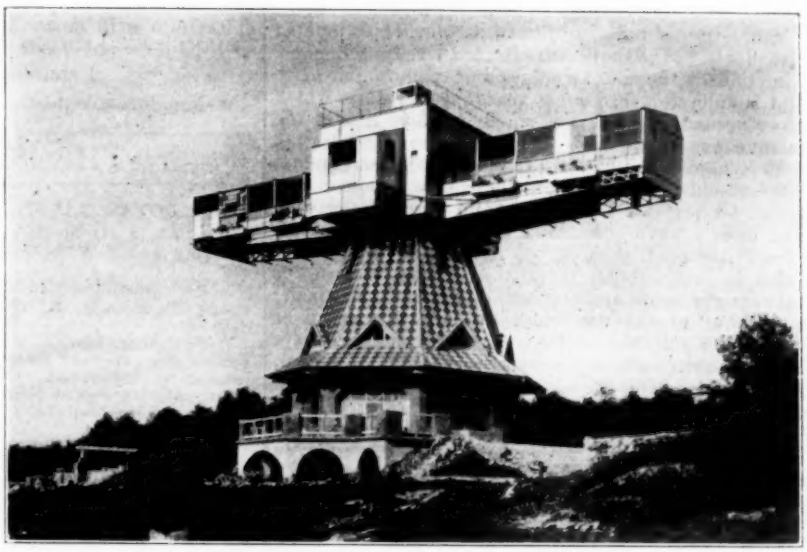
A visit to the solarium at Aix should be of great interest to those practising heliotherapy, for there is no doubt that with the principles adopted the best use can be made of sunlight treatment.

I am, Sir, yours faithfully,

HERMAN S. N. MENKO.

Northwick-terrace, N.W., August 8th, 1931.

FIG. 1.



View of the solarium.

FIG. 2

Part of a treatment room.

ADMINISTRATION OF CALCIUM.

To the Editor of THE LANCET.

SIR,—In his note on Intramuscular Injections of Calcium in your issue of August 22nd Dr. Izod Bennett recommends the use of a solution of calcium gluconate and lævulinate. For some years past in cases which have needed calcium and where for any reason the intravenous route has been deemed inadvisable, I have used injections of calcium gluconate (Sandoz) intramuscularly. In several hundred injections which have been given I have had no case of necrosis. The dose I have commonly used has been 10 c.cm. of a 10 per cent. solution. I have often found it of value to combine this therapy with massive doses of calcium gluconate and lactose on an empty stomach; 1 drachm of calcium gluconate and 1 drachm of lactose in a suitable vehicle is of great value and there is no doubt, as many recent workers have shown, that lactose assists the absorption of calcium. I have not found the oral administration of calcium very helpful in the small doses which are often suggested. In cases of chilblains, urticaria, hay-fever, and even in the parkinsonism following encephalitis lethargica I have had good results by these methods. I believe—and I have had some radiological evidence to confirm this—that the small hard tablets of calcium preparations sometimes pass unaltered through the intestine.

I am, Sir, yours faithfully,

Harley-street, W., August 27th, 1931. GERALD SLOT.

FISSURES AT THE ANGLES OF THE MOUTH.

To the Editor of THE LANCET.

SIR,—In the excellent paper by Dr. Daniel T. Davies on simple achlorhydric anæmia, published in THE LANCET on August 22nd, mention is made of cracks and fissures at the angles of the mouth as occurring in this disease. I have noticed this physical sign for many years in patients suffering from all sorts of diseases, and sometimes none at all, who all had one thing in common—viz., large artificial dentures. And it is the daily taking out and putting in of these plates which cause and keep up the cracks, where this is done carelessly. After the patient has been advised to exercise great care in manipulating the plates so as not to overstretch the mouth, the cracks soon heal.

I am, Sir, yours faithfully,

Leeds, August 30th, 1931. HERBERT J. ROPER.

SYPHILIS AND YAWS.

To the Editor of THE LANCET.

SIR,—I have no wish to prolong this correspondence, but I should like to point out the microscopical differences between syphilis and yaws. These seem to me to settle the whole question. W. Fox (*Brit. Jour. Ven. Dis.*, 1928, iv., 68) came to the conclusion that the histology was a complete answer to Hutchinson. J. M. H. Macleod (*Brit. Med. Jour.*, Sept 21st, 1901; Practical Handbook of the Pathology of the Skin, London, p. 200) sets out these differences. In yaws the cellular infiltration is not clustered round the vessels, as in syphilis, and there are no multinuclear cells, giant cells, or intracellular hyaline degeneration as in yaws. So far as the fibrous stroma is concerned, rarefraction of collagen is more marked in yaws, and in that disease there is no organisation or colloid degeneration such as occurs in gummata. The vessels in yaws show no proliferative changes in walls and endothelium as do those in syphilis. In yaws there is a marked proliferation and downgrowth of epithelium with great thickening of the horny layer, such as does not occur in syphilis.

I am, Sir, yours faithfully,

Municipal Clinic, Salford. E. TYTLER BURKE.

THE SERVICES

ROYAL NAVAL MEDICAL SERVICE.

Surg. Lts. A. D. Sinclair to *Centurion*; R. M. B. Lowis to *Moth*; and J. W. L. Crosfill to *Foxey*.

ROYAL NAVAL VOLUNTEER RESERVE.

Surg. Lt.-Commr. H. Winstanley to *Victory* for Haslar Hospital.

Proby. Surg. Lts. J. F. Heggie and F. T. Land to *Victory* for Haslar Hospital; G. E. W. W. Bayly, entered as Proby. Lt., and attached to List 1, Severn Divn., outlying unit at Newport.

Proby. Surg. Sub-Lt. B. R. Alderson to *Pembroke*, for R.N. Barracks.

Surg. Sub-Lt. A. P. Trimble to be Surg. Lt.

Surg. Sub-Lt. J. C. Moor to be Surg. Lt.

ROYAL ARMY MEDICAL CORPS.

ARMY RESERVE OF OFFICERS.

Lt.-Col. N. D. Walker has attained age limit; Capt. A. L. Taylor, from Supp. Res. of Off., to be Capt.

R. L. H. Minchin to be Lt.

ARMY DENTAL CORPS.

Lt. A. F. D. Shapland resigns his commn.

TERRITORIAL ARMY.

Edward Davies-Thomas to be Lt.

INDIAN MEDICAL SERVICE.

Lt. J. H. Boultbee to be Capt. (prov.).

The promotion of Maj. R. A. Warters is antedated to July 12th, 1927.

The undermentioned officers retire: Lt.-Col. A. G. Tresidder and Maj. R. R. M. Porter, on account of ill-health.

A SOUTH-WESTERN CRIPPLES' TRAINING CENTRE.—Under the auspices of the Plymouth and district cripples' aid committee it is proposed to open in Devonshire a training college for cripples to serve the whole of the south-western counties. At the end of March there were 567 cases under the care of the committee.

FINANCES OF THE BIRMINGHAM HOSPITALS.—An appeal is shortly to be launched for £90,000 on behalf of the General Hospital (£50,000), the Queen's (£30,000), and the Ear and Throat (£10,000). The Maternity Hospital has already made an appeal for £15,000, and the Skin and Urinary Hospital will in the near future ask for the means of increasing the number of its beds from 22 to 50; the Children's Hospital is likewise contemplating an appeal. The General Hospital will have an overdraft of £40,000 by the end of September, and the bank is asking for its reduction, £25,000 of it being unsecured. The number of beds for paying patients is about to be doubled. The Queen's has an unsecured overdraft of £22,000, and all its free investments have been sold to meet the additional maintenance cost of the new surgical block of 120 beds. Last year the Ear and Throat Hospital spent £3303 in excess of its income, £2000 worth of investments have been sold this year, and there is still an overdraft of £3500. The city's hospitals now contain a total of 1734 beds; in 1930 there were 28,825 new in-patients and 190,467 new out-patients. Meanwhile the Lord Mayor, as President, with other officials of the Birmingham Hospital Saturday Fund, which forms an integral part of the contributory scheme, has issued a circular setting out the case for raising the weekly contributions of members from 2*d.* to 3*d.* It is pointed out that although this fund is the most successful in the world it is inadequate and not self-supporting, and that the contributory patients are costing the hospitals many thousands a year more than they are paying for their treatment. The proposal has been considered by the board of delegates of the Saturday Fund.

PUBLIC HEALTH SERVICES

ANNUAL REPORTS OF MEDICAL OFFICERS OF HEALTH.

THE following are 1930 statistics of three counties :—

Name of district and estimated mid-year population.	Birth-rate.	Death-rates per 1000 of the population.					Death-rates per 1000 births.		
		All causes.	Tuberculosis.	Cancer.	Respiratory diseases with influenza.	Heart disease.	Infant mortality.	Young child (1–5 yr.) mortality.	Maternal mortality from childbirth.
Derby-shire .. (624,300)*	16·9	10·1	0·59	1·12	1·3	1·9	61	—	4·1
Surrey .. .. (861,850)*	15·2	10·0	0·62	1·52	1·0	2·1	41	15	3·4
Hampshire .. (432,600)*	16·5	11·3	0·70	1·62	1·1	2·5	44	20	3·8

Registered stillbirths per 100 live births: Derby-shire, 4·6, Surrey, 3·3; Hampshire, 3·3.

* The 1929 populations have been used.

Derbyshire.

Dr. W. M. Ash describes how the new duties of vaccination, infant life protection, and the maintenance of institutions for the physically and mentally sick, placed on the county council by the Local Government Act, 1929, are being dealt with. There are 19 vaccination officers and 81 public vaccinators and the contracts with them have been renewed excepting for the portions of their districts which are without the county boundary. The duty of supervising infants under 7 years of age who are received for reward has been met by appointing the county health visitors as infant life protection visitors. These children numbered 80 at the end of the year. Since the work was transferred from the guardians, parents have been requested, in four cases, to find more suitable foster-parents and in one case a child has been removed from unsuitable guardians by order. The maternity and child welfare committee also supervises the children boarded out by the public assistance committee. It has not been possible for the health committee to take over any of the poor-law hospitals, seeing that only two "institutions" permit of efficient separation of the hospital from the "house," and that there is no superfluity of beds which would allow the institutions to be closed for poor-law purposes. The health committee has not yet consulted the representatives of the voluntary hospitals with a view to formulation of a complete scheme. There has been no important change in the administration of out-relief since the transfer, but all assistance since April 1st has been given to the blind under the Act of 1920. Dr. Ash shows in this, as in previous reports, how an efficient and adequate general hospital system can be built up for the county and he hopes that two hospital services, voluntary and municipal respectively, will not be set up unless they are run in close coöperation and avoid unnecessary competition. He advocates one comprehensive institution for each area as a centre for preventive and curative medicine. The public assistance institutions are not, he says, suitable for the accommodation of many young and middle-aged people suffering from chronic disease and new provision is required for such.

The county mental hospital at Mickleover in its 24 wards has accommodation for 388 males and 382 females, and during the year a new up-to-date admission block for 100 patients has been opened, which is claimed to be one of the most complete buildings of this type. The county council has no mental deficiency institution and this is a pressing need. At the end of 1930 there were 30 male and 115 female mental defectives accommodated in certified institutions outside the county. The county council scheme for dealing with the 15 isolation hospitals proposes to diminish their number and to have three isolation hospital areas. The formulated principles of the scheme appeared to be generally acceptable to the local authorities, but when it came to deciding as to which hospitals should be closed it was immediately apparent that most of the local authorities qualified their acceptance of the scheme by a proviso that their own particular hospital should not be closed. Dr. Ash points out that the present isolation hospital system does not cover the whole county, does not attract an efficient staff, and does not uniformly adopt immunisation methods against diphtheria and scarlet fever. Further it provides, as a rule, for diphtheria and scarlet fever only save, sometimes, for typhoid fever. There is no provision for the proper nursing of measles and no accommodation for cerebro-spinal fever, encephalitis, dysentery, or poliomyelitis. The provision for small-pox Dr. Ash describes as lamentable, as many of the small-pox hospitals are not reasonably fit for human habitation. He wishes to see a small and a large hospital unit established in each of the three isolation hospital areas for dealing with small-pox. Occasional cases would be dealt with at the small unit and the larger units would be used for other county purposes during inter-epidemic periods. The accommodation for tuberculosis patients was improved during the year by the opening of a new 32-bed pavilion for adults of both sexes suffering from surgical tuberculosis. There is a lack of beds for advanced pulmonary cases, and Dr. Ash suggests that when the isolation hospital service is reorganised the small isolation hospitals, which are closed, could be readily adapted for observation and advanced cases of pulmonary tuberculosis and equipped with modern X ray apparatus. In this manner the needed additional beds for advanced and observation cases could be provided in situations accessible to relatives whilst freeing the sanatorium from the type of case which should not be there.

The county council has two maternity homes and contracts for the use of beds at four others. Each of the eight public assistance institutions also provides maternity beds but frequently in a very wasteful manner. For example, the county maternity home at Ashbourne is by no means fully used but the county council provides a maternity ward at its Ashbourne public assistance institution to which only one case was admitted during the year. A similar state of affairs exists at Chesterfield. The county council contracts for admission of both mother and child to the Royal Infirmary, Derby, in cases of ophthalmia neonatorum. During the year 65 cases were notified, of which 51 were treated at home and 14 in hospital. Vision was impaired in two cases. The antenatal scheme conducted by Miss E. E. Stephens, M.D., at 12 clinics throughout the county has grown rapidly since its inauguration in 1928. The attendances numbered 3117, as compared with 2316 in the previous year. During the year a child guidance clinic was opened at Matlock and is conducted by a member of the medical staff who has had special experience. Children are seen by appointment only.

Of 457 samples of sewage effluents 261 were classified as good, 119 as satisfactory, 48 as unsatisfactory, and 29 as bad. The county council has laid down a standard for the production of clean milk and during 1930 the two county sanitary inspectors visited a large number of cowsheds with the local sanitary inspectors and gave advice, which was well received. The entries for the clean milk competitions show an

increase and the inspecting judge reported very favourably on the high standard of efficiency attained.

Surrey.

Dr. J. Ferguson says that before April 1st, 1930, there had been in Surrey for 100 years the whole or large portions of 12 poor-law unions, and that the former boards of guardians interpreted the term "destitute" in a wide sense. The county council is anxious to transfer the care of the sick to the public health committee, of the mentally deficient to the mental hospitals committee, and the education of children to the education committee; and the public assistance committee is endeavouring to overcome the difficulties arising from the fact that few of the former hospitals are administered separately from the rest of the institution. The county council has, however, decided to appropriate the Chertsey and Bletchingly institutions for the care of mental defectives. There are in all 10 institutions with 1918 beds and the largest hospital, that of Kingston and district (547) beds) is that which can be most easily adapted for separate use and transferred to the health committee. Towards the end of 1929 the Surrey voluntary hospitals consultative committee, consisting of 24 members representing the governing bodies and medical staffs of the voluntary hospitals, was constituted. At its meetings this committee has made valuable constructive criticisms of the proposals placed before it by the county council. The voluntary hospitals have 1277 beds, the isolation hospitals 633, and the mental hospitals 2507. As regards medical out-relief there has been no change in the policy pursued by the former boards of guardians.

The county health visitors have been approved as infant life protection visitors. On April 1st there were 214 foster mothers who received 304 children, and at the end of the year 406 foster-mothers had given notice in respect of 501 children. The boroughs of Guildford and Wimbledon applied to the Ministry and were appointed local supervising authorities under the Midwives Acts, but a similar application by four urban district councils was not approved. In 1929 a whole-time woman medical officer with special experience in midwifery, &c., was appointed to undertake antenatal work and, at the 15 special clinics throughout the county conducted by her, 977 women attended antenatally and 132 postnatally. The Surrey Council has contracted with the L.C.C. for the institutional treatment of ophthalmia neonatorum. Notifications numbered 46. The cases in the practice of midwives numbered 26, of which 22 were treated at home and 4 in hospital. There were two deaths and vision was unimpaired in the remainder. The result in the 20 cases which occurred in the practice of doctors is not given.

The county council is making arrangements to obtain particulars of the housing situation from the rural districts. On the basis of four persons per house new provision was made for housing 44,000 persons and provision for 22,056 was in hand at the end of the year, but 1436 completed houses and 1288 in course of construction are L.C.C. houses to accommodate residents in the County of London. Mr. F. W. Medlock, M.R.C.V.S., and his four assistants have carried out a quarterly inspection of the herds of the Surrey cowkeepers. The cow examined numbered 67,443, of which 59,415 were in milk. The number of animals dealt with under the Tuberculosis Order was 142, 41 of which were reported to the police as suspect and 101 of which were detected by routine inspection. At the end of 1930 the river Wandle improvement works were practically completed.

In addition to participating in the London V.D. scheme the Surrey Council has clinics at Guildford and Redhill, at both of which the attendances increase. An additional session became necessary at Guildford where there is an ad hoc clinic in the hospital grounds. It is hoped soon to have a third clinic at Kingston and District Hospital. The 14 isolation hospitals have been surveyed and a scheme for the provision of adequate accommodation for the county is in course of preparation. For small-pox the Whitmoor Hospital (10 beds) makes provision for Godalming, Woking, and the urban and rural districts of Guildford, whilst the Clandon Hospital (37 beds) provides for Croydon and the rest of the county. During 1930, 40 cases were admitted to Clandon and the hospital was only closed for eight days during the year. Two of the cases were laundresses infected in the course of their duties. In May to July there was a considerable outbreak of paratyphoid in Barnes—49 cases, of which 16 were males and 33 females. Children and young persons were specially attacked. Fifteen of the patients were severely ill but there were no deaths. The cases were widely distributed over the area and no definite cause for the outbreak was discovered.

Hampshire.

Dr. H. Leslie Cronk reports that up to the end of 1929 grants of £13,761 and loans of £1148 had been paid on 156 cottages under the Housing (Rural Workers) Act, 1926, and that all but six of the 22 rural districts had made applications. Up to the end of 1930 grants totalled £23,231 and loans £2870 with respect to 297 cottages, showing that the Act was more used in 1930 than in any previous year. The extension of the Act and the further provision of £7000 for loans and grants by the county council in February, 1931, will allow further use to be made of this Act, which, says Dr. Cronk, alone enables the preservation of old cottages, that are picturesque assets to the county and can be let at a lesser rental than any new cottage. Under the Housing Act of 1930 a questionaire was issued by the housing sub-committee to obtain information with regard to the housing position in the 22 rural districts. This was followed by a conference in February, 1931, at which 19 of the rural district councils were represented. Exact information was not forthcoming as to how many of the 4087 subsidised houses erected since 1926 were for the agricultural population. Dr. Cronk thinks the problems for the rural districts are provision for housing shortage and the renovation of unfit houses, and that the latter problem can best be dealt with under the Housing (Rural Workers) Act. For any scheme to relieve shortage the county council would have to be satisfied that the houses were needed for the agricultural population.

The accommodation available for the sick in public assistance institutions and the best means of coöperation with the voluntary hospitals are now under consideration. Particulars of the 16 isolation hospitals in the county are given, but nothing can be done in the matter of eliminating unsatisfactory small hospitals and the concentration of beds in as few centres as possible until extensive alterations in the local sanitary areas have been arranged. Small-pox is accommodated at Winchester and at Basingstoke, and plans have been prepared for the improvement of the Winchester hospital.

During 1930 there were 18 antenatal clinics at work which were attended by 412 expectant mothers. In January, 1931, 31 antenatal clinics were established and on May 31st there were 34 in operation, 10 staffed by an assistant county medical officer, and 24 by practitioners of the neighbourhood with special experience. The new cases during the first quarter of 1931 were 382, as compared with 412 during the whole of 1930. Transit is frequently a difficulty for the women and the county council has agreed to pay the fees of general practitioners for domiciliary antenatal visits in such cases. Expectant mothers found antenatally to require dental treatment are treated by the council's dental surgeons. Of 107 mothers needing treatment 97 were actually treated during 1930. The premium to be paid by expectant mothers to ensure against the cost of medical attendance has been raised from 5*s.* to 7*s.* 6*d.* in first pregnancies, but in spite of this the number of insurances has increased. During 1930, 3079 midwives' cases insured and to them a doctor was summoned in 40 per cent. at a net cost to the council of 5*s.* 2*d.*

per case; whilst there were 2336 uninsured midwives' cases, to which the doctor was called on 833 occasions at a net cost to the council of 5*s.* 8*d.* The changes in the reasons for summoning medical aid, says Dr. Cronk, show that more attention is being paid by midwives to the necessity for calling in the doctor for complications which really matter. In the 19 public assistance institutions for which the county council is responsible, 47 beds are allocated for maternity cases, but only 102 births took place in these institutions during the year. The council's health visitors took over infant life protection work on April 1st. It was found necessary to withdraw approval from four foster-mothers formerly supervised by the guardians and from two foster-mothers notified after April 1st. Of 14 applications to receive foster-children two had to be refused as unsuitable.

The fifth county clean milk competition attracted a largely increased number of competitors. A new system has been introduced by which those who have attained the required standard in previous competitions are excluded from competing but become eligible for a Register of Accredited Milk Producers. Of the number eligible, 34 decided to register and both surprise and routine samples will be taken of their milk to ensure that they continue to practise what they have learned in the competitions. There is no routine inspection of the herds, and for checking the sale of tuberculous milk reliance is placed on notification by the owner, on outside complaints of tuberculous milk, on reports of tuberculous carcasses from the county slaughter-houses, and on the biological examination of 800 samples of milk supplied by the district councils. These methods resulted in 43 cows being dealt with under the Order.

INFECTIOUS DISEASE

IN ENGLAND AND WALES DURING THE WEEK ENDED AUGUST 22ND, 1931.

Notifications.—The following cases of infectious disease were notified during the week:—Small-pox, 21 (last week, 19); scarlet fever, 1200; diphtheria, 652; enteric fever, 52; pneumonia, 393; puerperal fever, 39; puerperal pyrexia, 136; cerebro-spinal fever, 29; acute poliomyelitis, 8; acute polio-encephalitis, 2; encephalitis lethargica, 10; dysentery, 5; ophthalmia neonatorum, 110. No case of cholera, plague, or typhus fever was notified during the week.

The number of cases in the Infectious Hospitals of the London County Council on August 25th-26th was as follows: Small-pox, 27 under treatment (last week 35 under treatment, 2 under observation); scarlet fever, 1087; diphtheria, 1368; enteric fever, 18; measles, 134; whooping-cough, 362; puerperal fever, 22 (plus 14 babies); encephalitis lethargica, 211; poliomyelitis, 7; "other diseases," 106. At St. Margaret's Hospital there were 12 babies (plus 5 mothers) with ophthalmia neonatorum.

Deaths.—In the aggregate of great towns, including London, there was no death from small-pox, 4 (1) from enteric fever, 13 (0) from measles, 3 (1) from scarlet fever, 11 (3) from whooping-cough, 18 (0) from diphtheria, 37 (6) from diarrhœa and enteritis under two years, and 17 (1) from influenza. The figures in parentheses are those for London itself.

The fatal cases of enteric fever outside London occurred, two at Portsmouth and one at Nottingham. Three deaths from measles were reported from Leeds, two from Bradford. Hull reported four deaths from diphtheria. Diarrhœa was credited with eight deaths at Liverpool, four at Birmingham, and three at Newcastle-on-Tyne.

The number of stillbirths notified during the week was 315 (corresponding to a rate of 46 per 1000 births), including 52 in London.

ENLARGEMENT OF DUDLEY GUEST HOSPITAL. The nurses' home of this institution is to be enlarged immediately to accommodate 20 additional nurses. A further 28 beds will then be available, bringing the total to 135.

MEDICAL NEWS

Royal College of Physicians of Edinburgh.

On the occasion of the 250th anniversary of the College it has been decided to confer the honorary Fellowship on Sir David Bruce, Lord Dawson of Penn, Sir Archibald Garrod, Sir William Hale-White, Dr. Robert Hutchison, Sir Thomas Lewis, Sir Donald MacAlister, Sir Edward Sharpey-Schafer, Sir Charles Scott Sherrington, Dr. Charles Achard (Paris), Dr. Frederick Grant Banting (Toronto), Dr. A. A. Hijmans van den Bergh (Utrecht), Dr. Knud Faber (Copenhagen), Dr. George Richards Minot (Boston, U.S.A.), Dr. Friedrich von Müller (Munich), Dr. Hermann Sahli (Berne), Dr. William Sydney Thayer (Baltimore), and Dr. Julius Wagner-Jauregg (Vienna).

Medical Society of London.

The programme for the first half of the 159th session opens with the annual general meeting at 8 P.M. on Monday, Oct. 12th, after which at 8.30 P.M. Mr. Herbert Tilley will give his presidential address on the Laryngoscope in Medicine. Subjects for the fortnightly Monday evening discussions are as follows: Oct. 26th, Blood Transfusion, to be introduced by Prof. Alex. Fleming, Dr. H. L. Tidy, and Mr. Geoffrey Keynes. Nov. 23rd, Methods of Determining Liver Function and Their Value, to be introduced by Sir Humphry Rolleston and Prof. E. C. Dodds. Dec. 14th, X Rays in the Diagnosis of Thoracic Disease, to be introduced by Dr. P. J. Kerley, Dr. F. G. Chandler, and Mr. J. E. H. Roberts. All these discussions begin at 8.30 P.M. The clinical evening on Nov. 9th begins at 8 P.M.

Epsom College.

A vacancy has arisen for a Salomons entrance scholarship of £50 a year. Candidates must be sons of legally qualified members of the medical profession either living or deceased, and must have been over 11 and under 14 years of age on Jan. 1st last. They must show an adequate standard of education for their age, and their financial condition must be such as to make it impossible to obtain an education at the College without the help of the scholarship. Applications, in proper form, must reach the Secretary at 49 Bedford-square, London, W.C.1, by the morning of Sept. 30th.

Instruction for D.P.H.

A three months' course of lectures and demonstrations on clinical practice and in hospital administration will be given at the North-Western Hospital, Lawn-road, Hampstead, N.W.3, by Dr. Alexander Joe, the medical superintendent, on Mondays and Wednesdays at 3.30 P.M. and alternate Saturdays at 11 A.M., beginning on Wednesday, Sept. 30th. Application should be made to the Medical Officer of Health, London County Council, Public Health Department (Special Hospitals), Victoria Embankment, E.C.4.

Middlesex Hospital Medical School.

The ninety-seventh winter session will open on Thursday, Oct. 1st, at 3 P.M., in the Queen's Hall, Langham-place, London, W., when Prof. James McIntosh will deliver the introductory address and Sir John Bland-Sutton will distribute prizes gained during the previous year. At 7.30 P.M. on the same day the annual dinner will be held at the Savoy Hotel.

National Smoke Abatement Society.

The third annual conference of this society will meet at Liverpool from Sept. 18th to 20th, under the presidency of Dr. H. A. Des Voeux. Among the speakers will be Dr. J. Johnstone Jervis and Dr. James Bennett.

Fellowship of Medicine and Post-Graduate Medical Association.

There will be six special courses in September at the following institutions:—(1) Hospital for Consumption and Diseases of the Chest, Brompton, from Sept. 7th to 12th, all day. (2) Bethlem Royal Hospital, Monks Orchard, Eden Park, Beckenham, from Sept. 8th to Oct. 3rd, on Tuesdays and Saturdays at 11 A.M.; lecture-demonstrations in psychological medicine. (3) Infants Hospital, Vincent-square, from Sept. 14th to 26th, from 2 P.M. daily, with special visits in addition; course in diseases of infants. (4) Central London Ophthalmic Hospital, Judd-street, from Sept. 15th to Oct. 8th, each afternoon; lecture-demonstrations in ophthalmology. (5) Westminster Hospital, from Sept. 14th to 26th, all day; course in general medicine and surgery, for men only. (6) Metropolitan Hospital, Kingsland-road, from Sept. 28th to Oct. 10th, all day; course in general medicine and surgery. Copies of all syllabuses may be had from the Fellowship at 1, Wimpole-street, London, W. 1.

Society of Medical Officers of Health.

The Neech prize, given for the best paper read at a meeting of this society during the session 1929–30, has been awarded to Dr. G. C. M. M'Gonigle of Stockton-on-Tees, for his address on The Biological Concept of Preventive Medicine, delivered before the Northern Branch and published in *Public Health* for May, 1930.

Paddington Medical Society.

The next meeting of this society will be held at 9 P.M. on Tuesday, Sept. 8th, at the Great Western Royal Hotel. Dr. J. D. Kidd will deliver an address on Dental Abnormalities and their Influence on General Disease.

Tropical Hygiene and Nursing.

A series of lectures and demonstrations on this subject will be given at 9, Chesham-street, London, S.W.1, on Monday, Wednesday, and Friday afternoons from Sept. 28th. Inquiries should be made to the secretary of the County of London branch of the British Red Cross Society at that address.

St. Mark's Hospital.

This City-road hospital for cancer, fistula, &c., last year treated 872 in-patients, and 1901 new out-patients. There were 859 operations, and during the year there were 29 deaths. With an income of £15,587, there was a credit balance of £26. Investigations are being made into the cause and treatment of cancer, in alliance with the British Empire Cancer Campaign. The Williand Shepherd estate has allocated £2500 for the provision of an electric lift.

Eversfield Chest Hospital.

Lord Brentford last week opened an annexe to this hospital for consumption and diseases of the chest at St. Leonards, which has cost £6000. The Railway Mission convalescent home, near the hospital, has been purchased and equipped, and it is proposed to transfer to it the women patients, the existing wards being reserved for men. The number of beds will now be increased to 100, and some small wards will be available for private patients. The hospital was founded by Dr. Thomas Gambier in 1884, and will shortly be celebrating its jubilee. It was transferred to its present site, on land given by Mr. C. G. Eversfield, in 1891, and then opened with 53 beds. Standing on a cliff 115 feet above the sea, it has a direct southern aspect, but avoids the "close" heat of the sea-shore. The committee is able to show a favourable financial report for the past year, but funds are urgently needed to meet expenditure on enlargement.

Contributory Schemes in Manchester and Merseyside.

For some time past the Manchester and district hospital councils, representing all the voluntary hospitals in the area, have been considering a scheme for rationalising the finances of these institutions. Representatives met in Manchester on August 19th and discussed the establishment of contributory schemes whereby a small weekly sum would be payable on a flat-rate basis. There is already in Manchester a contributory scheme, but it is regarded as not extensive enough. Under a scheme drawn up by Mr. Thomas Denman, a London accountant, it is suggested that the flat-rate which all work-people might pay during good health and employment for the provision of hospital accommodation and other sickness benefits, should be 3*d*. a week. The contributions to the scheme should be pooled. The total number of potential users of hospitals in Manchester and Salford is estimated at 916,000, and the total for East Lancashire at about 3,305,000. It is expected that at the end of five years at least £120,000 a year would be received from 200,000 contributors in Manchester and Salford and £400,000 a year from 700,000 contributors in the districts outside. By way of preliminary, an attempt is to be made to bring together representatives of the Manchester Hospital Saturday Fund and the Hospital Sunday Fund to see if the two organisations can work together with a saving in efficiency.

In connexion with the Manchester proposals, it may be noted that the Merseyside Hospitals Committee controls a penny-in-the-pound fund, whereby the ordinary income in Liverpool's voluntary hospitals was raised from £226,518 in 1927, when the committee was instituted, to £269,325 in 1930—an increase of £48,807. The employed have paid a penny a week for every pound of their wages (deducted at the source) and the employers have added another third. To-day there are more than a quarter of a million contributors. The sum raised in 1928 was £18,000 and last year £126,420. It is stated that at least three-quarters of a million people on Merseyside are assured of free hospital treatment.

Mr. Neville Chamberlain, the Minister of Health, has appointed Mr. A. Nevil Rucker to be his private secretary, and Mr. W. H. Howes to be his assistant private secretary.

Royal Dental Hospital of London.

The prize distribution and conversazione of this hospital will be held on Friday, Oct. 2nd, at 8 P.M., when Prof. Elliot Smith, F.R.S., will preside.

Charing Cross Hospital.

Last year there were 4797 in-patients, whose average stay was 20 days, and whose average cost was £11 8*s*. 3*d*. There were 28,784 new out-patients, 19,630 of whom were casualty cases; they cost 7*s*. 6*d*. each. The income was £63,624, and the expenditure £64,467.

Solvent Hospitals.

For the year ending on June 30th last, Bishop Auckland cottage hospital had a credit balance of £283; last year Brampton war memorial cottage hospital, Cumberland, spent £26 less than its income. Newcastle-on-Tyne Dental Hospital closed last year with a credit balance of £640.

Brompton Hospital for Consumption.

Last year this hospital admitted 2276 new in-patients, 305 of whom were transferred to the Frimley sanatorium. There were 4502 new out-patients. The income was £76,902, which left a credit balance of a little over £1000. The largest item of receipts was £37,400, payments by or on behalf of patients; voluntary gifts amounted to £12,144. The work of reconstruction is now complete, and the accommodation for paying patients is greatly appreciated.

Provincial Radium Institute.

The annual report of the Manchester Radium Institute states that the work "is very seriously affected through the lack of funds with which to maintain the necessary equipment and staff to cope with the increasing demands for radium treatment." There was an adverse balance of £2283, but despite financial difficulties, the work continues to grow. The surgeons have performed 303 operations within the Institute and 800 in other hospitals in the district. The radiologists examined 1260 patients, and found that 758 were suitable for treatment; during the year 655 of them were actually treated.—The North of England Council of the British Empire Cancer Campaign has sanctioned a grant of three-fourths of the estimated cost—£20,000—of founding an institute at Newcastle-on-Tyne Infirmary.

Medical Diary

Information to be included in this column should reach us in proper form on Tuesday, and cannot appear if it reaches us later than the first post on Wednesday morning.

LECTURES, ADDRESSES, DEMONSTRATIONS, &c.

FELLOWSHIP OF MEDICINE AND POST-GRADUATE MEDICAL ASSOCIATION, 1, Wimpole-street, W.

MONDAY, September 7th, to SATURDAY, September 12th.—HOSPITAL FOR CONSUMPTION, Brompton, S.W.3. All-day course in Diseases of the Chest—lectures, demonstrations, and operations. Fee £3 3*s*.

ST. MARK'S HOSPITAL, City-road, E.C.

THURSDAY, Sept. 10th.—4.30 P.M., Mr. W. B. Gabriel: The Elucidation of Rectal Symptoms. 1. Bleeding.

THE LANCET: SUBSCRIPTION RATES.

INLAND	One Year	£2	2	0
	Six Months	1	1	0
	Three Months	0	10	6
ABROAD	One Year	2	10	0
	Six Months	1	5	0
	Three Months	0	12	6

SMALL ADVERTISEMENT RATES.

Books and Publications .. .. .. } Four lines
Official and General Announcements .. } and under 6*s*. 0*d*.
Trade and Miscellaneous Advertisements }

Every additional line 1*s*. 6*d*.

For complete scale of advertisement charges apply to THE MANAGER.

Vacancies

For further information refer to the Advertisement columns.

Albert Dock Hospital, Connaught-road, E.—Res. M.O. At rate of £110.

All Saints' Hospital for Genito-Urinary Diseases, 91, Finchley-road, N.W.—H.S. At rate of £150.

Altrincham General Hospital.—Jun. H.S. At rate of £120.

Bedford County Hospital.—Asst. H.S. At rate of £130.

Birmingham and Midland Eye Hospital, Church-street.—H.S. £110.

Birmingham City Education Committee.—Special Schools M.O. and Director of Child Guidance Clinic, not exceeding £800.

Birmingham General Hospital.—Res. Anæsthetist. £120. Also 2 H.S.'s. Each £70.

Birmingham, Monyhull Colony.—Second Asst. M.O. £350.

Blackburn County Borough.—Asst. School M.O., &c. £600.

Brighton Sanatorium and Infectious Disease Hospital.—Jun. Res. M.O. At rate of £150.

Bristol City and County Mental Hospital.—Fourth Asst. M.O. £350.

Bristol Eye Hospital.—Anæsthetist. Also Res. H.S. £150.

Carnarvonshire C.C.—Deputy M.O.H. and Asst. School M.O. £700.

Cambridge, Addenbrooke's Hospital.—Two H.S.'s. At rate of £130.

Cancer Hospital, Fulham-road, S.W.—H.S. At rate of £100.

Croydon General Hospital.—Hon. Surgeon for Ear, Nose, and Throat Dept.

Cumberland Infirmary, Carlisle.—H.P., Second H.S., H.S. to Special Depts. All at rate of £155.

Derby, Derbyshire Hospital for Women, Friar Gate.—H.S. At rate of £150.

Dreadnought Hospital, Greenwich.—H.P. and H.S. Each at rate of £110.

Dudley, Guest Hospital.—H.S. £170–£200.

East Ham Memorial Hospital, Shrewsbury-road, E.—H.P. At rate of £150. Also H.S. to Special Depts. and Cas. O. Each at rate of £120.

Evelina Hospital for Children, Southwark, S.E.—H.P. At rate of £120.

General Lying-in Hospital, York-road, Lambeth, S.E.—Jun. Res. M.O. and Anæsthetist. At rate of £100.

Great Yarmouth General Hospital.—H.S. £140.

Harrogate General Hospital.—H.P. At rate of £150.

Harrogate Infirmary.—Jun. H.S. At rate of £125.

Harrow-on-the-Hill, Harrow and Wealdstone Hospital.—Res. M.O. At rate of 140 gns.

Hastings, Royal East Sussex Hospital.—Sen. H.S. At rate of £150.

Hereford, Herefordshire General Hospital.—H.S. £150.

Hull, Corporation of Kingston-upon-Hull and Hull and Goole Port Sanitary Authority.—Asst. M.O.H. £600.

Ilford, King George Hospital.—H.S. At rate of £100.

Leeds City.—Chief Asst. M.O.H. £900.

Liverpool Eye and Ear Infirmary, Myrtle-street.—H.S. to Ophth. Dept. £120.

Liverpool, Mill-road Infirmary.—Res. Asst. M.O. £200.

Longton Hospital, Staffs.—H.S. £150.

Macclesfield General Infirmary.—Res. H.S. £180.

Manchester Royal Children's Hospital, Pendlebury.—Two Asst M.O.'s for Out-patients' Dept. Each at rate of £150. Also Res. M.O. At rate of £125.

Manchester Royal Eye Hospital.—Jun. H.S. £120.

Manchester Royal Infirmary.—H.S. for Central Branch, Roby-street. At rate of £100.

Manchester and Salford Hospital for Skin Diseases.—H.S. At rate of £100.

Margate Royal Sea Bathing Hospital for Surgical Tuberculosis.—H.S. At rate of £200.

Margate and District General Hospital.—Res. M.O. £150.

Miller General Hospital, Greenwich-road, S.E.—Cas. O. and H.P. At rate of £150 and £125 respectively.

Newcastle-upon-Tyne Hospital for Sick Children.—H.P. and Sen. and Jun. H.S.'s. All at rate of £100.

Northampton General Hospital.—H.P. and Four H.S.'s. Each at rate of £150. Also Res. Dresser. £10 for six months.

Nottingham General Dispensary, Broad-street.—Res. Surg. £250.

Oldham, Boundary Park Municipal Hospital.—Res. Asst. M.O. £200.

Plymouth, South Devon and East Cornwall Hospital.—Res. Anæsthetist and H.S. At rate of £100.

Port Said, British Hospital.—Res. Asst.M.O. £375.

Princess Beatrice Hospital, 194, Finborough-road, S.W.—R.M.O.

Princess Louise Kensington Hospital for Children, St. Quintin-avenue, Kensington, W.—H.P. At rate of £100.

Queen's Hospital for Children, Hackney-road, E.—Asst. Surgeon. Also H.P. At rate of £100.

Reading, Royal Berkshire Hospital.—Radiological H.S. and Res. Anæsthetist. At rate of £150.

Richmond Royal Hospital, Surrey.—Jun. H.S. At rate of £100.

Royal Eye Hospital, St. George's Circus, S.E.—Twelve salaried Refractionists, twelve Clin. Assts., and Pathologist.

Royal National Orthopædic Hospital, 234, Great Portland-street, W.—Two H.S.'s. Each at rate of £150. Also H.S. for County Branch, Brockley Hill, Stanmore. At rate of £150.

Royal Northern Hospital, Holloway, N.—H.S. At rate of £70.

Royal Waterloo Hospital for Children and Women, Waterloo-road, S.E.—Hon. Clin. Asst.

St. Peter's Hospital for Stone, &c., Henrietta-street, Covent Garden, W.C.—H.S. At rate of £75.

Salisbury, General Infirmary.—H.P. £150.

Sheffield, Jessop Hospital for Women.—Sen. Res. M.O. £200. Also Res. M.O. and two Asst. H.S.'s. At rate of £150 and £100 respectively.

Sheffield Royal Hospital.—Ophth. H.S. Also Anæsthetist. £120 and £80 respectively.

Sheffield Royal Infirmary.—Asst. Aural and Ophth. H.S. and H.S. and Second Asst. Cas. O. Each at rate of £80.

South Eastern Hospital for Children, Sydenham, S.E.—Asst. Res. M.O. At rate of £100.

Stafford, Staffordshire C.C. County Bacteriological Laboratory.—Asst. Bacteriologist and Pathologist. £700.

Stockport Infirmary.—H.P. £175.

Swansea General and Eye Hospital.—H.P. £150.

Torquay, Torbay Hospital.—H.P. and H.S. Each £175.

University College Hospital, Gower-street, W.C.—Clin. Pathologist. £750.

Watford, Herts., Peace Memorial Hospital.—Res. and Surg. M.O.'s. Each at rate of £150.

Weir Hospital, Grove-road, Balham, S.W.—Res. M.O. £150.

West London Hospital, Hammersmith-road, W.—Res. Asst. S. £200. Also H.P., H.S.'s, and Res. Anæsthetist. All at rate of £100.

Westminster Hospital, Broad Sanctuary, S.W.—H.S. to Ear, Nose, and Throat Dept. At rate of £102.

Weston-super-Mare General Hospital.—Res. H.S. £150.

Wigan Royal Infirmary.—H.S. At rate of £150.

Winchester, Royal Hampshire County Hospital.—H.S. At rate of £100.

Windsor, King Edward VII. Hospital.—H.S. At rate of £100.

Wolverhampton, Royal Hospital.—H.S. for Orthopædic Dept. At rate of £100.

Woolwich and District War Memorial Hospital, Shooters Hill, S.E. Res. M.O. £175.

Worcester County and City Mental Hospital, Powick.—Jun. Asst. M.O. £350.

The Chief Inspector of Factories announces the following vacant appointments for Certifying Factory Surgeons: at Wolston (Warwick); Milnthorpe (Westmorland); and Ulceby (Lincs.).

The Home Secretary gives notice that in consequence of the death of Dr. H. MacIntyre, of Bridgend, Islay, one of the Medical Referees under the Workmen's Compensation Act, for part of the Sheriffdom of Argyll, he is considering the appointment of an additional Medical Referee. Applications for the post should be addressed to the Private Secretary, Scottish Office, Whitehall, London, S.W.1, and should reach him not later than Sept. 24th, 1931.

Births, Marriages, and Deaths

BIRTHS.

AVERY.—On August 27th, the wife of Harris Avery, M.D., Selsdon-road, S.E., of a daughter.

BOUSFIELD.—On August 25th, at Cheyne Lodge, Denmark Hill, the wife of Guy Bousfield, M.D. Lond., of a son.

DEVEREUX.—On August 24th, at St. Monica's, Kirkley Cliffe, Lowestoft, the wife of C. H. Devereux, M.B., B.S. Lond., of a son.

FRANCIS.—On August 22nd, at Chiaran, Guildford, the wife of C. A. Francis, M.B., Ch.B. Camb., of a daughter.

MILLER.—On August 29th, in Edinburgh, the wife of Dr. R. H. Miller, West African Medical Staff, of a daughter.

NIXON.—On August 30th, at 7, Lansdown-place, Clifton, Bristol, to Doreen, wife of Dr. J. A. Nixon—a son.

STAFFORD.—On August 30th, at Hayes-crescent, Golders Green, the wife of Major H. Neville Stafford, *M.C.*, R.A.M.C., of a son.

MARRIAGES.

BUXTON—WARLOW.—On August 31st, in London, St. John Dudley Buxton, F.R.C.S., second son of the late Dr. Dudley Buxton, to Winifred, younger daughter of G. Picton-Warlow, L.D.S. Edin., of Birkenhead.

GRANT—GAMBLE.—On August 8th, at the Chapel of the Warwick County Mental Hospital, Hatton, Arthur Stanley Grant to Florence Margaret Gamble, M.D. Lond., M.R.C.P.

KERR—KEAY.—On Sept. 1st, at St. George's Presbyterian Church, Southport, Dr. James R. Kerr, C.B.E., to Helen Dishington, daughter of Mr. and Mrs. A. D. Keay.

DEATHS.

BURGESS.—On August 26th, at a London nursing home, James George Burgess, L.S.A., of Upwell, Wisbech, aged 76 years.

HARDY.—On August 24th, at a nursing home, Parkstone, Leonard Ernest Hardy, M.B.C.M. Edin., F.R.C.S.I., aged 65.

LYDALL.—On August 20th, at Briars Hey, Stechford, Birmingham, after a short illness, Wykeham Tracy Lydall, M.D. Brux., M.R.C.S., L.R.C.P. Lond., aged 59.

MACLEHOSE.—On August 30th, at Shona, Ayr, Norman M. MacLehose, M.B., C.M. Glasg., D.P.H. Camb., of Cranley-place, S.W.

MILLN.—On August 28th, Surgeon-Captain J. D. S. Milln, M.B., Ch.B. Edin., R.N., retired, Alverstoke, Hants.

PRICE.—On August 29th, at Klosterlea, Reading, John Alfred Parry Price, M.D. Oxf.

REED-HILL.—On August 28th, at Hangleton-road, Brighton, very suddenly, William Reed-Hill, M.B., C.M. Edin., aged 75.

WHITTICK.—On August 30th, at Sherwood-court, W., Fallon Percy Whittick, M.D. Durh.

N.B.—A fee of 7s. 6d. is charged for the insertion of Notices of Births, Marriages and Deaths.

NOTES, COMMENTS, AND ABSTRACTS

THE DEATH OF SHAKESPEARE.

A SUMMARY.

By MACLEOD YEARSLEY, F.R.C.S.

THE Rt. Hon. J. M. Robertson, in "The Genuine in Shakespeare,"[1] alludes more than once to the elusiveness of the greatest of the world's poets: "Alike in the biographical details that reach us," he says, "and in the inferences we can draw from the work done in the closing years, Shakespeare remains elusive to the last." In the manner of his death he seems none the less elusive, and its cause has been made the subject of almost as much fantastic memoir as his work. It may be of interest to review the varied opinions which have been advanced, and to consider what there is in the way of evidence upon which to base deductions as to fact.

First and foremost is the will, from which it appears (1) that the draft was made on or shortly before Jan. 25th, 1616 (n.s.); (2) that two months later this rough draft was signed in three places by Shakespeare on his death-bed, March being substituted for January; and (3) that four weeks later he died. The handwriting of the poet comes next in importance. Six undisputed signatures exist, all dated—an important point—between 1612 and 1616, and in all of which various persons have discovered traces of "nervous disease." The three pages of the play of *Sir Thomas More*, now in the British Museum, are still the subject of dispute as to the genuineness of the caligraphy as Shakespeare's; Sir E. Maunde Thomson[2] contending for its genuine nature on fairly good grounds, Dr. S. A. Tannenbaum[3] disputing his verdict. There remain besides a number of signatures, some of which are known to be spurious, some are very strongly suspect, while a very few *may* be genuine. Dr. Tannenbaum seems to hold an unduly suspicious attitude towards the latter, sweeping them away as forgeries in so arbitrary a manner as almost to suggest an obsession.

Ward, the Vicar of Stratford who kept a diary from 1648 to 1679, must have been in an excellent position to obtain the signature pasted in the second folio which belonged to him, but this Dr. Tannenbaum contemptuously repudiates as spurious. This folio is now in the United States; it surely should be in Stratford. It has always seemed to me, however, that some of these signatures might conceivably be genuine, and that to reject them after comparison with those which are undisputed and, according to experts, show signs of disease, and which date within four years of Shakespeare's death, is alike unscientific and unfair. The experts who have condemned these six autographs as tremulous and faulty seem to forget that the earlier signatures of a man who "never blotted a line" must show a difference, and that one who writes much and quickly may slur words, and especially names, in forming them.

Thirdly, there are local traditions, chiefly represented by that of Ward in his diary, to the effect that "Shakespeare, Drayton, and Ben Jonson had a merie meeting, and itt seems drank too hard, for Shakespeare died of a feavour there contracted." Whether this is true or a mere fragment of distorted gossip is doubtful; it may be worthy only to be ranked with the declamation over killing a calf and the deer-stealing traditions.

Aubrey's remark that when, latterly, men would have him join in drinking bouts, he would refuse, saying "he was in pain," seems to bear a genuine stamp. I am not aware that eyestrain has ever been suggested for this "pain," but headache due to that condition might well have been the reason.

A great deal of stress has been laid—unduly, in my opinion—upon Shakespeare's retirement to Stratford in 1610, at the, as some writers have put it, "comparatively early age" of 46. It is not known in what sense he retired, but at least he was near the end of his working period. Whether 46 was then deemed an early age may be debated, but when the late fifties seem to have been accounted as "venerable eld" it may have been regarded as middle-age, and middle-age would certainly be an excellent time for retiring, especially to one who had made a fair fortune and loved the country as Shakespeare loved it. Therefore it is injudicious, in the absence of more definite evidence, to rely too much upon this "early" retirement as indicative of "incipient paralysis" or other serious disorder. What is known of Shakespeare's purchases and general conduct in those last years at Stratford do not suggest any mental impairment.

Nisbet, in his remarkable "Insanity of Genius," devotes the whole of Chapter VI. to Shakespeare and his family, and, as is too often the case with an author who has a pet theory to prove, labours to show that the Master Poet did not escape the common lot that Nisbet gives to geniuses. He recites the "extraordinary mortality" of Shakespeare's brothers and sisters, and gives a table from which he deduces that the stock was clearly not a healthy one, the average life being less than 32 years. But this includes a sister, Anne, who died at 8, and the youngest brother, Edmund, of whom Nisbet "guesses" that he succumbed, at 27, to consumption. He suggests that neuropathic conditions would account for such a family history, pointing out that through Joan alone (who lived to 77) was the Shakespeare blood transmitted beyond the third generation. Gilbert, who came next to the poet and died at 46, he considers was "probably" a hypochondriac.

The theory that Shakespeare's death resulted from fever Nisbet cannot accept, even in the form of typhus or typhoid. The insanitary conditions of an Elizabethan county town, however, is extremely likely to have resulted in a disease of that nature. Nisbet points out that an attack of dangerous illness which caused the hurried signing of the will, followed by an interval of four weeks before death, is not the course of typhus, but he misses the fact that it might well fit in with mild, "ambulatory" typhoid, and such an explanation would appear to be one which might account for death from "a feavour" following the indiscretion mentioned by Ward.

The testamentary signatures Nisbet considers as showing, in conjunction with indications of failing memory in the drawing up of the will, "beyond a doubt the nature of Shakespeare's last illness." His "condition when he penned these signatures must have been one of extreme nervous agitation." What, Nisbet asks, was the nature of this prostration of the nervous system? and suggests "a paralytic or epileptic seizure," a precursory evidence of which is shown in the 1612–13 signatures. Nisbet's argument is too insistent and his postulation of the nature of the illness is too vague.

Dr. Tannenbaum[3] (p. 76) makes short work of Nisbet's arguments as "wholly unwarranted guesses," and illogical deductions. He also—and rightly—refuses the conjectures of Dr. A. Guthmann[4] that Shakespeare died the victim of arterio-sclerosis resulting from mental overwork, chronic nicotianism, chronic alcoholism, gluttony, and sexual excesses. The second suggestion may fairly be refuted by the entire absence in the poet's works of any mention of tobacco. Indeed, there is not a shred of evidence for any of these charges, save that of mental overwork. Tannenbaum seems to think angina pectoris a likely cause (which would account for Aubrey's statement as to pain), or possibly "a severe heart attack which may have been followed by a pulmonary congestion which terminated fatally after four weeks," adding, anent Ward's story, that "gossip among the poet's Puritan neighbours would naturally fasten on the merry meeting as the cause of death."

Nor can he find the slightest justification for the guesses that he died of typhoid fever, of Bright's disease, or of apoplexy.

The late Dr. R. W. Leftwich, in a lecture before the Royal Society of Medicine on March 19th, 1919, considers that an examination of the six undisputed signatures furnishes unimpeachable evidence that Shakespeare suffered from Scrivener's palsy, as they show every one of the 18 classical symptoms of that disorder. Considering the enormous amount that Shakespeare must have written in the cramped Gothic style which preceded and for some time accompanied the later Italian hand, it might well be that he suffered from writer's cramp. Incidentally, such a continuous output would result in headache from eyestrain in a period when astigmatism went uncorrected. But people do not die of Scrivener's palsy. Dr. Leftwich can find no case to be made out for other diseases, and, as a medical man, he must be accounted as a competent witness. Like Dr. Tannenbaum, however, there may be with Dr. Leftwich a strain of the obsession of the expert graphologist. As Nisbet, himself not free from bias, wisely remarks, "Graphology contains no doubt a substratum of truth, though it may not yield results as definite as those its votaries claim for it." Dr. Leftwich states that "it has been surmised that the defects of Shakespeare's handwriting were due to some constitutional disease such as locomotor ataxy or chronic paralysis, but the preamble to his will, written only a few months before his death, negatives such views." Despite the looseness of this statement one must agree with it, and it may be pointed out in support that in the Elizabethan literature, dramatic and fictional, no allusions are to be found to a disease so prominent in its symptoms as locomotor ataxy, and that the nerve manifestations of syphilis were not the subject of notice before the eighteenth century.

From the above short review it is seen that Shakespeare's death has been attributed to a miscellaneous assortment of maladies. Fever, typhus, typhoid, paralysis, epilepsy, apoplexy, arterio-sclerosis, over-smoking, chronic alcoholism, gluttony, sexual excesses, angina pectoris, Bright's disease, pulmonary congestion, locomotor ataxy; a sufficiently heterogeneous list with which to saddle a man!

The hypotheses of medical criticism as to the poet's death appear to have attributed as many burdens to him as the literary critics have found for his work! Of the whole list, the most suggestive is that of ambulatory typhoid, but, as in the life, so in the death of Shakespeare nothing is certain. His elusiveness remains. He has not even left us a swan-song.

REFERENCES.

1. Routledge, 1930.
2. Shakespeare's Handwriting, Oxford, 1916.
3. Problems in Shakespeare's Penmanship, New York, 1927.
4. Shakespeare Jahrbuch, Leipzig, 1925, pp. 89–93.

FIRE INQUESTS.

A RECENT annual report of the Society for the Protection of Ancient Buildings warns the public of fire dangers to which our fine old country houses are exposed. In this connexion the report mentions a communication from Dr. F. J. Waldo in which the City coroner reiterates the need of technical equipment against fire and mentions his unique jurisdiction (under the City of London Fire Inquests Act of 1888) for investigating fires in the City. Dr. Waldo has more than once suggested that what is good enough for the City of London might well be good enough for the rest of the country; but proposals for the extension of the fire inquest system outside London, though favoured some 20 years ago, are nowadays neglected. Fire inquests could claim an ancient and unbroken pedigree. According to the Mirror of Justices a treatise ascribed to the reign of Edward I., coroners attended on occasions of arson and inquired who put the fire there and how and by what felony, drunkenness or other mischance the fire arose. Modern scholarship is contemptuous of the Mirror of Justices; Pollock and Maitland in their History of English Law decide to ignore its account of the criminal law as full of fables and falsehoods. Coke definitely says that coroners can inquire into no felony except homicide; Hale and Comyns agree with him. It is nevertheless on record that by local custom mediæval coroners inquired of other felonies in the turbulent county of Northumberland. In more recent times, according to Jervis on Coroners, inquests on non-fatal fires were held at Rotherham in 1843, at Doncaster in 1853 (on the burning of the parish church), and at Nottingham in 1857, as well as in other towns. Then in 1860 came the case of R. *v.* Herford wherein Chief Justice Cockburn and his colleagues reaffirmed the dicta of Coke, Hale, and Comyns. William Herford, coroner of Manchester, held an inquest on a fire which had occurred on the premises of a local shopkeeper, and, during an adjournment, the High Court intervened. Statutory confirmation of the limitation on coroners' jurisdiction was enacted in Section 44 of the Coroners Act of 1887. In the City of London fire inquests had been held from time to time though their legality was contested; the City of London Fire Inquests Act of 1888 regularised the practice.

More than once in the present century the extension of the practice to the whole country has been officially considered. In 1909 the Home Office departmental committee on the law of coroners examined the point at length. Evidence was heard from Dr. Waldo, who said that some 140 or 150 fires were referred to him every year; he made preliminary inquiries in all cases but held inquests in only a small percentage; he had never committed anyone for trial for arson. Fire inquests duties, he said, were more technical and exacting than body inquests; they took more time and required greater knowledge of the law. He thought the Act had caused a diminution of fires in the City and was useful in showing up neglect of precautions. On the other hand, the chief constable for Manchester deprecated any extension of the coroner's power; the ordinary law was enough: he claimed that there were fewer fires in Manchester than in the City of London. The committee's report (Cd. 5004) preferred Dr. Waldo's view. It declared that the City system had worked well and ought to be extended. The Act of 1888 was "preventive and remedial." The committee recommended that a beginning be made with an adoptive Act. County or borough councils might adopt the practice of fire inquests and, if the result was satisfactory, then the Act might be made universal. The recommendation made no more headway than Sir Henry Cotton's Fire Inquests Bill of 1909. In 1920 the London County Council inserted in a General Powers Bill a clause for the holding of such inquests in the county area (outside the City) on direction from the Council or the Home Secretary. The proposal was withdrawn, pending legislation for the whole of England, owing to disagreements about the coroner's initiative and remuneration.

The position was reviewed by the Royal Commission on Fire Brigades and Fire Prevention. Its report, published in 1923 (Cmd. 1945), remarked on the anomaly that Dr. Waldo should be able to hold fire inquests in the City but not in his adjacent jurisdiction of Southwark. The report stated that examination of fire outbreaks whether fatal or non-fatal would be "useful from several points of view." But the Royal Commission accepted an opinion pressed by the London County Council in evidence, that the tribunal should consist not of a coroner whose qualifications are usually medical and legal but of a specialist officer with technical knowledge of building construction, engineering, and fire prevention. The analogy of Board of Trade or Ministry of Transport inquiries into boiler explosions or railway accidents was evidently attractive. The Commission recommended that the Home Office should draw up a panel of persons thus qualified and that the new proposals should apply to Scotland. In Scotland at present the procurator fiscal can obtain an inquiry by the sheriff and a jury into any fatal accident, and in

towns the burgh prosecutor can investigate fire outbreaks if requested by the town council or on special report by the firemaster that wilful fire-raising is suspected. As matters thus stand, the Royal Commission's recommendations are an obstacle to the extension of the City of London practice. Indeed the ancient usefulness of the coroner as an investigator of crime is latterly somewhat belittled. Section 20 of the Coroners (Amendment) Act of 1926, which put the coroner out of action when once the examining justices have taken in hand proceedings in cases of suspected homicide, was a serious break with the past.

BRITISH DERMATOLOGY.

To the "Annals of Medical History" (Vol. III., New Series, July, 1931), Dr. Haldin-Davis contributes an article of real historic interest setting out the foundations of British dermatology, from which we learn how comparatively modern is anything like rational treatment of skin diseases. From the very dawn of medicine affections of the skin must have received attention from doctors, for the sufferers from skin diseases are more urgent than any class of patients in their demands for relief, while it is a lamentable fact that a substantial proportion of them can only be offered remedial measures of a partial or temporary character. But there is hopefulness in the fact that with the general development of pathology more and more dermatological defaults, following upon the discovery of their origin, are yielding to intelligent therapeutics. Dr. Haldin-Davis attributes the progress of the last century mainly to the publication of Robert Willan's treatise in 1808. This was an attempt at the rational classification of skin diseases based upon characteristic lesions, and the author completed half of the projected work during his life-time. His pupil, Thomas Bateman, completed the treatise which was published under the heading "A Practical Synopsis of Cutaneous Diseases, according to the Arrangement of Dr. Willan." The basis thus introduced into dermatological classification has largely remained; where possible the scientific procedure of classifying by ætiology has replaced Willan's classification by appearances; but, as Dr. Haldin-Davis points out, our knowledge of the ætiology of skin diseases is lamentably deficient at the present day, except in the case of a few infective agencies, compelling us largely to classify skin diseases according to their appearances. Reading this article it becomes clear that Willan was a very remarkable man, for without the microscopic aid by which so many advances in this field of medicine have been made, he devised a system of classification 130 years ago which to-day is the main medium for intelligent converse between experts.

BLOOD PRESSURE IN THE CAPILLARIES.

RESEARCH which produces important results is of obvious value, but just as useful is the work which repeats research in order to confirm or disprove it. This checking of results will often prevent a whole crop of errors.

For a long time attempts have been made to measure the pressure in the capillaries. Many different methods have been applied, but so far no satisfactory solution of the problem has been found. Recently A. Sokolowski and M. Kubiczek [1] have undertaken investigations into the method designed by Kylin—a method of compression, which has hitherto been accepted as giving the most accurate data. They approached the problem from two aspects, considering first the technical difficulties arising from imperfections in the apparatus, and secondly, the results as interpreted in the light of the anatomy and histology of the part investigated. The imperfections in the apparatus, they found, made comparative study impossible and by certain modifications they tried to overcome them. Then they succeeded in making several interesting observations. For example, they noted that after the initial compression the capillary loops continued to contract despite a rapid diminution in the pressure. This suggested an active contraction on the part of the vessels as a result of irritation of the vasomotor nerve-endings in the vessel walls; consequently, any results obtain with this apparatus would be influenced in great part by the vasomotor nerves. Again, when an attempt was made to get repeated readings for comparative study—as, for example, when testing the effects of certain drugs—it was found that after each compression a strong vascular hyperæmia ensued, necessitating a pause of four to six minutes between each reading.

The second half of the work consisted of a careful examination of the anatomy and minute histology of the part which had been investigated—namely, the skin just behind the nail bed. Sections of various thicknesses were taken and stained by several methods. Correlating the results, they came to the conclusion that readings obtained by Kylin's method of compression, and, in fact, all compression methods, did not give the pressure in the capillaries, but gave actually the pressure in the afferent arterioles; in the area under examination the arteriole was the one described by and named after Spalteholz. Again, the results were influenced by the thickness of the skin of the subject investigated, and by several other factors.

The main result from these investigations is to show the innumerable difficulties in the way of any attempt to measure capillary pressure, and the inaccuracies of an accepted method.

[1] Ann. de Méd., 1931, xxix., 408.

ANÆMIA IN PREMATURE BABIES.

As a further supplement to the work of Dr. Helen Mackay [1] comes the report of some investigations by Dr. G. Sanpaolesi,[2] of Florence, carried out in Prof. Czerny's clinic in Berlin. He examined the blood of twelve premature babies at approximately weekly intervals, beginning from two days to three months after birth. He enumerated the total red blood corpuscles present, estimated the amount of hæmoglobin, and calculated the water content by the indirect method of determining the total solids. All the babies were born normally, ten were one month premature and two were two months premature. In one case congenital heart disease was present and another suffered from a congenital syphilitic infection. Otherwise the children were normal and developed normally except for slight digestive troubles which were without influence on the weight curve. None of the children showed evidence of the so-called exudative diathesis, held by some authorities to be the cause of the hydræmia from which they all appeared to suffer in the third month of life. In the first four weeks Sanpaolesi found normal values for total solids and water content of the blood but in the second month began a decrease in the solids and increase in the water content, associated with a parallel decrease in the number of red blood corpuscles and amount of hæmoglobin present. This was not accompanied by any alteration in the weight curves and in the child with congenital syphilis the decrease in total solids was more marked than in the normal babies, while the patient with congenital heart disease had only a minimum decrease. In two cases where vegetable additions to the diet were early introduced the decrease in total solids was very slight and there was a correspondingly quick recovery after the third month, although no influence of the diet on the total red cell count and hæmoglobin content could be detected. Sanpaolesi thinks that iron deficiency is not of great importance in the anæmia of the premature baby which seems to be due rather to a disturbance of the water-regulating mechanism of the body. Further observations on the total blood volume are necessary to settle this point and they present great practical difficulty. For the moment he merely draws the conclusion that there is certainly a dilution of the blood in the third month of life in premature babies and that this plays a part in producing what may be called the physiological anæmia of prematurity.

[1] See THE LANCET, July 25th, p. 195.
[2] Jahrb. f. Kinderh., 1931, cxxxii., 277.